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THE RELATIONSHIP OF THE ROENTGENOGRAPHIC APPEARANCE OF THE PULMONARY ARTERY TO PULMONARY HEMODYNAMICS*†

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INTRODUCTION

ALTERATIONS in the dynamics of pulmonary arterial blood flow occur in a wide variety of disorders of the heart, the lungs, and the great vessels. The pulmonary artery itself, however, is simply a channel for conducting blood from the right heart to the lungs. Thus, despite the complexities of the underlying disorders, alterations in this simple system are limited to abnormalities of pressure and flow, and intrinsic diseases of the artery. It is the purpose of this paper to determine the changes which are present in the pulmonary artery, as seen on routine roentgenographic and roentgenoscopic examinations, in known conditions of intrinsic disease and altered pressures and flows.

METHODS

Diagnoses of the cases used in this study were based on venous catheterization of the heart and the pulmonary artery.[1,2,3] Pressures in the pulmonary artery were recorded through the catheter by the method of Hamilton.[4] Pulmonary artery blood flows were determined by application of the Fick principle. Oxygen consumptions initially were calculated from regular hospital metabolic rates done just before catheterization. More recently, the consumption of oxygen has been derived from expired air collected in Douglas bags simultaneously with blood sampling, measured in a Tissot spirometer and analyzed for oxygen and carbon dioxide by the Haldane method. Blood samples collected under oil through the catheter from the pulmonary artery and from a systemic artery through an inlying needle were analyzed for oxygen by the method of Van Slyke and Neill.[5] The oxygen content of pulmonary venous blood was considered equivalent to that of systemic arterial blood provided there was no veno-arterial shunt. When such shunting did occur, the oxygen content of pulmonary venous blood was assumed to equal 98 per cent of the oxygen capacity.

In order to analyze the roentgenographic changes recognizable by routine methods, changes in size and activity of the main pulmonary artery were recorded. The term

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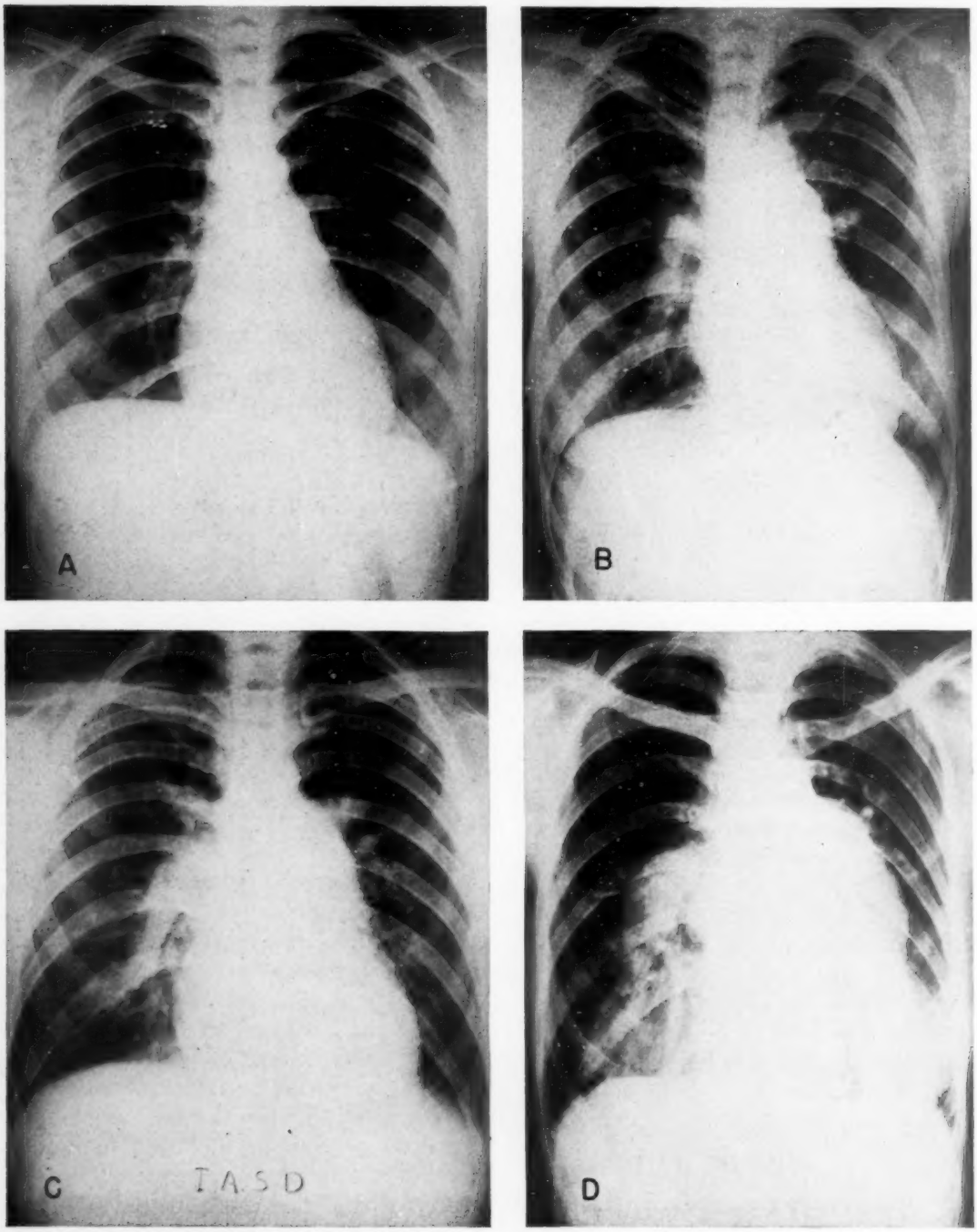

FIG. 1. (*A*) Roentgenogram to illustrate Grade N+ enlargement of the main pulmonary artery and its hilar and intrapulmonary branches. Because of the wide limits of normal pulmonary artery size, Grade N+ variations are considered as being at the upper limits of normal (Case 13, Table I).

(*B*) Roentgenogram to illustrate a Grade ++ enlargement of the main pulmonary artery and a Grade +++ enlargement of its hilar and intrapulmonary branches (Case 4, Table III).

(*C*) Roentgenogram to illustrate a Grade +++ enlargement of the main pulmonary artery and its hilar and intrapulmonary branches (Case 3, Table III).

(*D*) Roentgenogram to illustrate a Grade ++++ enlargement of the main pulmonary artery and its hilar branches with a Grade ++ enlargement of its intrapulmonary branches (Case 9, Table II).

"main pulmonary artery" refers to a localized convexity or bulge in the left heart border seen immediately beneath the aortic knob. Actually, this convexity is made up of the main pulmonary artery and the proximal portion of its left main branch.[6,7] Changes in the size and activity of the hilar branches of the artery were also recorded. Hyperactivity of the hilar branches refers to a systolic expansile pulsation of these structures often described as a hilar dance. Changes in the caliber of the intrapulmonary branches of the artery were recorded. To facilitate the interpretation of these changes, a system of grading them was established. Figure 1 illustrates what is meant by N+, ++, +++, and ++++ enlargement of the pulmonary artery and its branches. Inasmuch as the pulmonary artery normally varies greatly in size, particularly in young people, Grade N+ variations are probably best considered as being at the upper limits of normal. Figure 6*D* illustrates what is meant by a −1 decrease in pulmonary arterial size.

Grading of hyperactivity on the basis of roentgenoscopic observations is admittedly difficult. Grade + has been selected to refer to slight but definite hyperactivity and Grade ++ to refer to more marked changes.

Because this study was carried out in an adult hospital, it did not include infants and young children under nine years. This

TABLE I

THE EFFECT ON THE PULMONARY ARTERY OF A WIDE RANGE OF RESTING FLOWS AT ESSENTIALLY NORMAL PRESSURES

Case Number	Patient	Main Pulmonary Artery		Hilar Branches		Distal Pulmonary Branches —Size	Pulmonary Artery Flow— l/min.	Pulmonary Artery Flow Index l/min./M²	Pulmonary Artery Pressure Systolic/ Diastolic mm. Hg
		Size	Activity	Size	Activity				
				Atrial Septal Defect					
1	J.V.	+++	++	++	++	++	34.5	16.3	30/15
2	S.L.	+++	++	++	?	++	16.1	10.3	33/12
3	H.C.	+++	++	+++	++	+++	14.0	7.8	35/10
4	V.C.	++	++	++	?	++	11.0	7.1	32/18
5	B.K.	++	++	N	+	N	11.8	8.6	18/7
6	G.O.	++	N	N+	?	++	27.9	15.1	36/4
7	R.S.	N+	?	N+	?	N+	10.6	6.9	27/8
				Patent Ductus Arteriosus					
8	M.C.	N+	+	N+	?	++	10.9	8.5	36/12
9	M.O.	N to ++	?	N+	N	N+	8.0	4.6	22/10
10	E.W.	N+	+	N+	N	N+	9.3	4.8	22/10
11	E.C.	N+	+	N+	?	N	9.1	4.9	38/12
12	T.B.	N+	+	N	N	N	8.9	6.2	25/11
13	E.S.	N+	+	N+	N	N+	5.9	3.3	22/11
14	M.J.	N+	?	N+	?	N+	5.9	3.4	15*
15	E.F.	N+	+	N+	?	N	5.6	2.9	18*
16	H.S.	N+	+	N+	N	N	4.7	3.0	14*
				Ventricular Septal Defect					
17	J.H.	N+	N	++	N	++	8.1	9.8	29/12

* Indicates mean pressure.

TABLE II

THE EFFECT ON THE PULMONARY ARTERY OF INCREASED RESTING PRESSURES WITHOUT SIGNIFICANTLY INCREASED FLOWS

Case Number	Patient	Main Pulmonary Artery		Hilar Branches		Distal Pulmonary Branches —Size	Pulmonary Artery Flow— l/min.	Pulmonary Artery Flow Index l/min./M²	Pulmonary Artery Pressure Systolic/ Diastolic mm. Hg
		Size	Activity	Size	Activity				
Eisenmenger's Complex									
1	M.K.	+++	++	+++	++	++	4.3	3.1	117/54
2	D.K.	+++	+	++	?	++	4.4	2.6	172/54
3	E.B.	+++	?	+++	?	++	7.2	4.4	115/60
4	S.G.	+++	++	++	?	N+	1.0	1.2	96/60
5	C.S.	+++	+	++	+	+++	7.2	4.0	124/64
6	A.G.	++	N	N+	N	++	5.1	3.6	128/81
7	E.G.	N+	N	N+	N	++	3.5	2.2	117/56
8	B.K.	++	N+	++	N	++	3.4	2.3	122/65
Atrial Defect with Pulmonary Vascular Disease									
9*	C.R.	++++	++	++++	++	++	3.0	1.9	80/30
Mitral Stenosis									
10	L.G.	++++	++	++	N	N+	5.4	3.3	97/58
11	M.G.	+++	N	+++	N	++	2.7	2.0	114/58
12	D.V.	+++	N	+++	N	++	3.0	2.7	84/45
13	H.H.	++	N	++	N	N+	4.4	2.2	( )
14	J.M.	++	N	+++	N	+++	4.4	2.6	40/16
15	K.O.	++	N	++	N	++	4.9	3.4	118/60
16	J.F.	N	N	N+	N	++	6.5	3.8	40/21
17	J.D.	N	N	N	N	N+	9.4	5.2	37/17
Pulmonary Vascular Disease									
18	L.S.	+++	+	++	+	++	5.0	2.9	87/35
19*	M.Q.	+++	N	N+	N	N+	3.0	2.0	80/55
20*	D.L.	+++	+	++	++	++	3.5	2.5	61/31
21	B.M.	++	N	N+	N	N+	8.1	4.4	59/31
22	R.V.	N+	N	N+	N	N	5.9	3.4	54/30

* Indicates autopsied case.
( ) Pressure not recorded, markedly elevated.

fact should be borne in mind in assessing the conclusions to be drawn.

OBSERVATIONS

1. The effect on the pulmonary artery of a wide range of resting flows at essentially normal pressure. The results of a study of 17 cases showing a wide range of pulmonary arterial flow at essentially normal pressures at rest are summarized in Table 1. Of the 17 cases, 7 had defects in the atrial septum, 1 had a defect in the ventricular septum, and 9 had patency of the ductus arteriosus. Each of these conditions permits shunting of oxygenated blood either directly or indirectly into the pulmonary artery. The artery, therefore, must carry not only its normal complement of blood, but also the additional amount shunted into it.

Table 1 demonstrates a rather striking correlation between the magnitude of the flow through a pulmonary artery and the recorded size and hyperactivity of that structure. With one major exception, the larger the flow was, the larger and more hyperactive were the main pulmonary artery and its branches. Case 6 was the exception in that despite a very large flow, the main pulmonary artery and its branches were only moderately dilated.

Viewing the results in Table 1 from a different point of view, it is apparent that of the 9 cases which showed flow indices of less than 7 L/min./M², only 1 (Case 9) showed an increase in size of the main pulmonary artery.

On the basis of this study, it would appear that small increases in pulmonary arterial flow rarely produce significant changes in the artery. Larger increases in flow (more than 7 L/min./M²) usually produce enlargement and hyperactivity that are proportional to the size of the flow. These points may be further emphasized

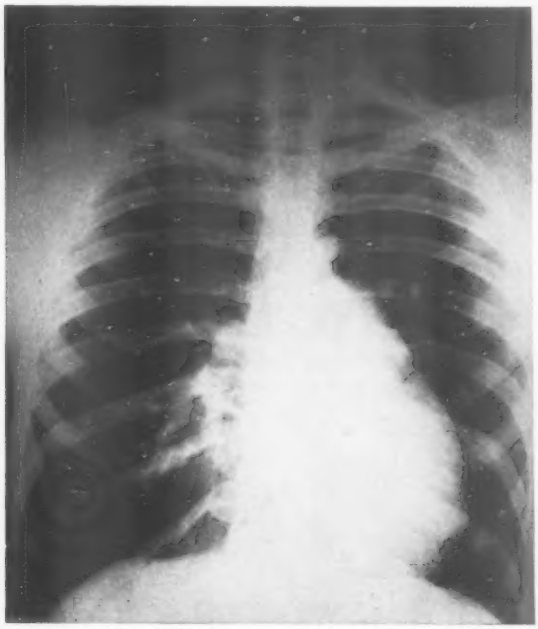

Fig. 2. Increased resting pulmonary artery flow. Case 1, Table 1. J. V., a twenty-one year old veteran of amphibious troop operations was discovered to have a heart murmur at examination for re-enlistment. Cardiac catheter study showed an atrial defect which permitted a huge pulmonary blood flow at normal pulmonary arterial pressures. The roentgenogram shows a Grade +++ enlargement of the main pulmonary artery and its hilar and peripheral branches. Roentgenoscopy revealed marked hyperactive expansile pulsations of the main pulmonary artery and of its hilar branches (hilar dance). Note the size of the pulmonary artery in this case as compared with that in Case 13 (Fig. 1*A*) where despite the presence of a patent ductus arteriosus the pulmonary artery flow was not large.

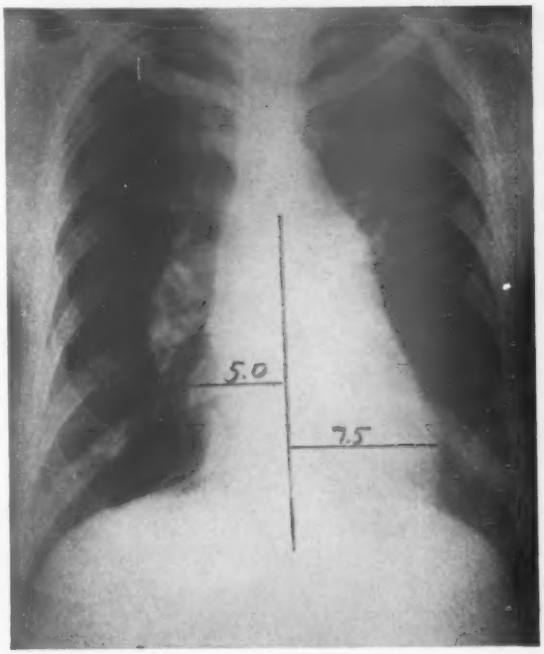

Fig. 3. Increased resting pulmonary artery pressure. Case 3, Table II. E. B., a thirty-two year old mother, known to have been cyanotic at birth, and limited by dyspnea and fatigue. The digits were clubbed. There was a moderately loud, rough, systolic murmur and a blowing, diastolic murmur in the pulmonic area. The electrocardiogram was characteristic of right ventricular hypertrophy. Cardiac catheterization demonstrated equal systemic and pulmonic arterial pressures, physiologic evidence of an over-riding aorta, as well as small veno-arterial and arteriovenous shunts. The pulmonary blood flow was slightly increased. The roentgenogram shows a +++ enlargement of the main pulmonary artery and its hilar branches with a ++ enlargement of the intrapulmonary branches. Note that the appearance of the pulmonary artery in this case is exactly comparable to that in Figure 2, emphasizing that either increased resting pulmonary artery pressure or flow may produce identical changes.

by comparison of Figure 1*A* (Case 13) with Figure 2 (Case 1).

2. The effect on the pulmonary artery of increased resting pressures without significantly increased flow. Twenty-two cases that showed increased pulmonary arterial resting pressures with essentially normal flows are summarized in Table II. The recorded data demonstrate that increased pressure in the pulmonary artery resulted in an enlargement of that structure which was entirely comparable to that seen in patients with increased flow. This enlargement included not only the main pulmonary artery, but also its hilar and intrapulmonary branches. While hyperactivity was not quite so striking a feature of increased pressure as of increased flow, the fact remains that it may occur and be quite marked. We are unable to distinguish the effects of increased flow with normal pressures from increased pressure with normal flows using the routine roentgenographic techniques for study of the pulmonary artery. Comparison of Figures 2 and 3 illustrates this point.

Case 14 is unusual in that there was marked enlargement of the pulmonary artery with only a slight elevation of its pressure.

3. The combined effect of an abnormal increase of flow and pressure on the pulmonary artery. Eleven cases in which both flow and pressure were elevated in the pulmonary artery are summarized in Table III. These cases differed from those in Table I only in that they had a definite pulmonic hypertension associated with an increased flow.

Since both increased pressure and flow cause enlargement and hyperactivity of the pulmonary artery, it is not surprising to find in Table III the combination of the two producing such a result. Except for

TABLE III

THE EFFECT OF AN ABNORMAL INCREASE OF FLOW AND PRESSURE ON THE PULMONARY ARTERY

Case Number	Patient	Main Pulmonary Artery		Hilar Branches		Distal Pulmonary Branches —Size	Pulmonary Artery Flow— l/min.	Pulmonary Flow Index l/min./M²	Pulmonary Pressure Systolic/ Diastolic mm. Hg.
		Size	Activity	Size	Activity				
Atrial Septal Defect									
1	F.S.	+++	?	+++	++	++	7.5	5.0	83/38
2	R.J.	+++	++	+++	++	+++	28.9	17.4	65/40
3	S.W.	+++	++	+++	++	+++	30.5	19.3	41/21
4	E.Mc.	++	++	+++	N	+++	10.2	7.1	75/29
5	S.Y.	++	+	++	+	+++	12.8	10.5	46/17
Patent Ductus Arteriosus									
6	M.F.	++	?	+++	?	+++	17.0	9.0	60/44
7	M.H.	++	?	++	?	+++	8.8	10.5	48/25
8	S.I.	N+	?	++	?	+++	8.8	10.5	48/30
9	E.M.	N+	N	N+	N	N+	29.3	20.0	45/23
Ventricular Septal Defect									
10	H.B.	+++	++	++	++	+++	8.1	9.8	100/49
11	E.P.	+++	++	+++	+	+++	21.5	15.1	64/36

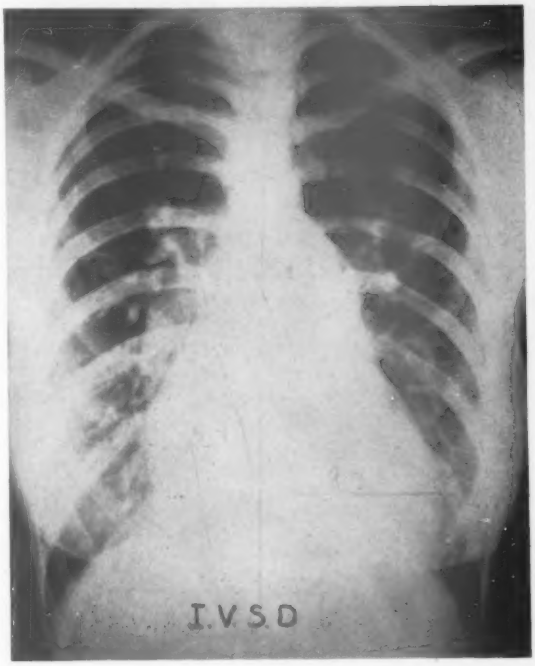

Fig. 4. Increased resting pulmonary artery pressure and flow.

Case 11, Table III. E. P., a twenty-two year old girl with known heart disease from the age of two and a half. Examination revealed a loud precordial systolic murmur with thrill. Right bundle branch block was present. Cardiac catheterization demonstrated an interventricular septal defect which permitted a very large blood flow through the pulmonary circuit, and a high pulmonary arterial pressure. The roentgenogram shows a Grade +++ enlargement of the main pulmonary artery and its hilar and intrapulmonary branches. Roentgenoscopy revealed moderate hyperactivity of the main pulmonary artery and expansile pulsations of the hilar branches. Comparison with Figures 2 and 3 demonstrates that the combination of increased resting pressure and flow in the pulmonary artery results in changes comparable to those of increased pressure or flow alone.

Case 1, flows alone were of sufficient magnitude to account for the recorded changes. In this case, however, the enlargement exceeded that expected from flow alone and probably resulted from the combined effects of both pressure and flow. If the present enlargement were due to a large shunt in the past—and such a shunt probably existed[8]—one would expect, on the basis of experience gained in the ligation of patent ducti, to have a diminution in size of the pulmonary artery along with a diminution of its flow.[8] Since this is not the case, it seems reasonable to conclude that both pressure and flow have been operating to produce the present picture. Figure 4 (Case 11) illustrates the changes seen in this group.

4. The effect on the pulmonary artery of pulmonic stenosis with normal flows distal to the stenosis. The 7 cases listed in Table IV were found to have pulmonic stenosis

Table IV

THE EFFECT ON THE PULMONARY ARTERY OF PULMONIC STENOSIS WITH NORMAL FLOWS DISTAL TO THE STENOSIS

Case Number	Patient	Main Pulmonary Artery		Hilar Branches		Distal Pulmonary Branches —Size	Pulmonary Artery Flow— l/min.	Pulmonary Artery Flow Index l/min./M	Pulmonary Artery Pressure Systolic/ Diastolic mm. Hg.	Right Ventricle Pressure Systolic/ Diastolic mm. Hg.
		Size	Activity	Size	Activity					
1	A.J.	+++	+	N	N	N	5.0	2.9	16/8	80/8
2	W.H.	+++	+	−N	N	−N	5.8	3.5	29/16	38/10
3	W.P.	++	++	N	N	N	7.2	4.6	23/6	39/4
4	T.M.	++	N	N	N	N	5.6	3.1	26/5	40/2
5	R.L.	N+	N	N	N	N	6.9	3.6	33/14	99/2
6	E.R.	N	N	N	N	N	5.3	3.4	22/15	35/6
7	J.Mc.*	N	N	N	N	N	2.8	1.6	28/10	65/0

* Calculated flow believed to be falsely low.

without an associated cardiac abnormality. Four of these 7 cases showed enlargement and definite hyperactivity of the main pulmonary artery. In a fifth case, the pulmonary artery was at the upper limits of normal in size. In none of these 7 cases, however, was there any enlargement or hyperactivity of the hilar or intrapulmonary branches. This dilatation, limited to the main pulmonary artery, seems to be a characteristic feature of pulmonic stenosis and usually distinguishes it from a simple increase in flow or pressure (see Table I, Case 5, and Table II, Case 19, for exceptions). It is not, however, an invariable feature because a perfectly definite pulmonic stenosis may exist without producing any recognizable change in the appearance of the pulmonary artery. This fact is amply illustrated by Cases 5, 6, and 7. As regards Case 7, it is believed that the pulmonary blood flow may well have been more normal than calculated and hence this case has been included in this table. The characteristic changes of pulmonic stenosis are seen in Figure 5 (Case 1).

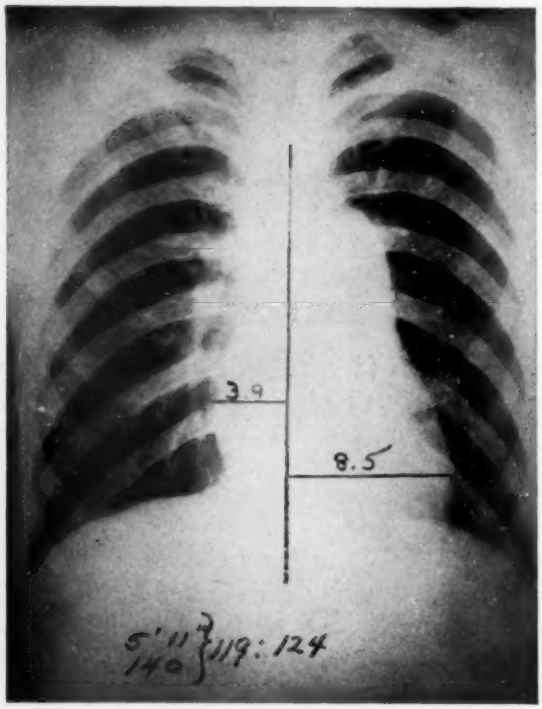

FIG. 5. Pulmonic stenosis with normal pulmonary artery flow.

Case 1, Table IV. A. J., a twenty-three year old male mechanic with heart trouble from the age of five, asymptomatic, but rejected for Army service because of an abnormal cardiac shadow. A loud pulmonic systolic murmur and thrill were present. The electrocardiogram showed right ventricular hypertrophy. A diagnosis of pulmonic stenosis was made on cardiac catheterization with the finding of a normal pulmonary artery pressure and an elevated right ventricular pressure. The blood flows were normal. The roentgenogram shows a Grade +++ enlargement of the main pulmonary artery, but no enlargement of the hilar or intrapulmonary branches. Roentgenoscopy showed hyperactivity of the main pulmonary artery but none of the hilar branches. Normal hilar and intrapulmonary branches distinguish this case from those illustrated in Figures 2, 3 and 4.

5. The effect on the pulmonary artery of pulmonic stenosis with reduced flows distal to the stenosis. The tetralogy of Fallot affords an opportunity to study the effects on the pulmonary artery of a reduced flow distal to a severe pulmonic stenosis. Along with the reduced flow, there is also a reduced pressure in the artery. The results of a study of 12 cases of the tetralogy of Fallot and of 1 case of severe stenosis without dextraposition of the aorta are summarized in Table V.

It has been thoroughly established that the pulmonary artery may be small in the tetralogy of Fallot,[10] and 3 of the cases in Table V showed this change, as illustrated in Figure 6*D*. Six of the cases, on the other hand, showed no deviation of the appearance of the pulmonary artery from the normal. Cases 1, 2, 3 and 4 showed rather unusual changes. The arteries of Cases 1 and 2 were exactly comparable to those seen in pulmonic stenosis with normal flow (see Fig. 6*A* and *B*). Case 1 was a severe pulmonic stenosis with a reduced cardiac output. In Case 2, because of the very small right-to-left shunt, the flow through the pulmonary circuit was essentially equivalent to that in the systemic circuit. In this respect this case resembled a severe pulmonic stenosis in which there was a reduced cardiac output. Case 3 (Fig. 6*C*) showed a localized saccular aneurysmal dilatation of the left pulmonary artery

and a moderate increase in the size of the intrapulmonary branches. No explanation can be given for these changes. Case 4 showed a small localized dilatation of the main pulmonary artery which did not resemble the changes of pulmonic stenosis. It should be emphasized that the patients in this study have survived to adulthood and cannot, therefore, be expected to resemble

2. Abnormal increases in resting flow of large size (greater than 7 L/min./M²) through the pulmonary artery usually produce changes which are roughly proportional to the size of the flow. These changes consist of an increase in the size and hyperactivity of the artery and its branches.

3. Abnormal increases in pressure in the pulmonary artery at rest result in changes

TABLE V

THE EFFECT ON THE PULMONARY ARTERY OF PULMONIC STENOSIS WITH REDUCED FLOWS DISTAL TO STENOSIS

Case Number	Patient	Main Pulmonary Artery		Hilar Branches		Distal Pulmonary Branches —Size	Pulmonary Artery Flow— l/min.	Pulmonary Artery Flow Index l/min./M²	Pulmonary Artery Pressure Systolic/ Diastolic mm. Hg.	Right Ventricle Pressure Systolic/ Diastolic mm. Hg.	Net Right-to-Left Shunt— l/min. mm. Hg.
		Size	Activity	Size	Activity						
Pulmonic Stenosis											
1	J.T.	+++	+	−N	N	−N	3.6	2.0	26/9	151/0	0.6
Tetralogy of Fallot											
2	S.K.	+++	+	−	N	−	2.1	2.2	10–20	110/8	0.4
3*	H.G.	N+	N	+++left N right	N	++	2.0	1.3	( )	122/10	1.8
4*	R.M.	N+	N	−	N	−	1.3	1.0	( )	( )	3.9
5	L.D.	N	N	N+	N	N	2.4	1.3	27/13	90/11	2.1
6*	P.H.	N	N	N	N	N	2.7	2.0	( )	97/8	1.8
7	E.E.	N	N	N	N	N	3.5	2.6	15/5	110/0	1.9
8	V.R.	N	N	N	N	N	2.3	1.5	18/8	140/9	2.4
9	D.M.	N	N	N	N	−	1.9	2.2	19/0	110/0	2.2
10	S.F.	−N	N	−N	N	−N	1.1	1.0	7	105/7	1.7
11*	R.D.	−	N	N+	N	N+	3.5	2.3	( )	93/6	3.6
12	A.D.	−	N	N	N	N	1.8	1.5	15	140/8	1.7
13	R.A.	−	N	−	N	−	2.4	2.1	18/9	124/8	1.9

* Pulmonary artery not entered.

entirely the severe forms of the tetralogy seen in early childhood.

SUMMARY AND CONCLUSIONS

An analysis has been made of the changes in the pulmonary artery recognizable by routine methods of roentgenography, in known conditions of altered pressure and flow at rest and in intrinsic disease. The results may be summarized as follows:

1. Abnormal increases in resting flow of moderate size (less than 7 L/min./M²) through the pulmonary artery rarely produce a recognizable change.

which are to us indistinguishable from those of increased flow.

4. The combination of an abnormal increase in both pressure and flow at rest produced changes that resemble those of either pressure or flow alone.

5. Pulmonic stenosis with normal pulmonary arterial flow and pressure is usually characterized by a poststenotic dilatation of the artery which in the cases studied was confined to the main pulmonary artery and did not include the hilar or the intrapulmonary branches.

6. Reduced flows beyond a pulmonic stenosis, seen best in the tetralogy of Fallot,

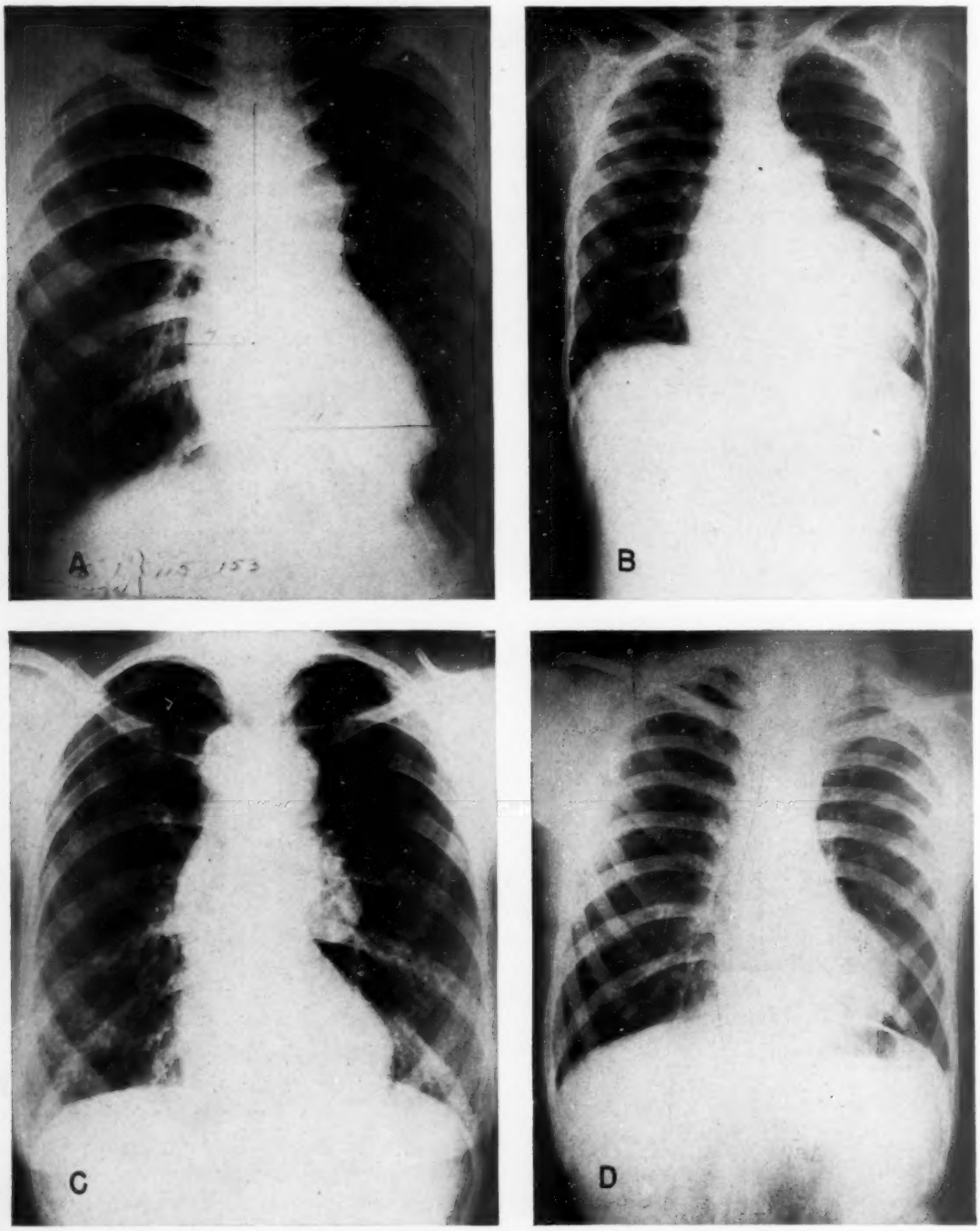

FIG. 6. Pulmonic stenosis with reduced pulmonary artery flow.

(*A*) Case 1, Table v. J. T., an eighteen year old student with known heart disease at birth. Dyspnea and cyanosis were present with exertion. Cardiac catheterization demonstrated a severe pulmonic stenosis with a reduced pulmonary arterial blood flow. There was arterial oxygen unsaturation due to a small right-to-left shunt. The roentgenogram shows a Grade +++ enlargement of the main pulmonary artery. The hilar and intrapulmonary branches are of less than normal caliber. Roentgenoscopy showed only slight hyperactivity of the main pulmonary artery.

(*B*) Case 2, Table v. S. K., a nine year old girl who developed bacterial endocarditis with pulmonary

often cause no recognizable change in the pulmonary artery. There may, however, be significant dilatation or reduction in the size of the main artery.

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emboli. There was slight digital clubbing and cyanosis. A widely transmitted precordial systolic murmur and thrill were present. The electrocardiogram showed marked right ventricular hypertrophy. Cardiac catheterization revealed a severe pulmonic stenosis with low pulmonary arterial pressure and reduced pulmonary blood flow. A small right-to-left shunt was present. The roentgenogram shows a Grade +++ enlargement of the main pulmonary artery with a reduction in the caliber of the hilar and intrapulmonary branches.

(*C*) Case 3, Table v. H. G., a thirty-six year old woman cyanotic from birth and troubled by dyspnea. The cardiac catheter passed directly into the aorta from the right ventricle. The blood flow through the lungs was low and there was moderate arterial oxygen unsaturation due to a moderate right-to-left shunt. The roentgenogram shows a double arched aorta, a saccular dilatation of the left hilum and an increase in caliber of the intrapulmonary branches. Roentgenoscopy gave no additional information.

(*D*) Case 13, Table v. R. A., a twelve year old boy with known heart disease and cyanosis since birth. A loud blowing systolic murmur was heard over the precordium. Catheterization findings were typical of the tetralogy of Fallot. There was a low pulmonary artery flow. The roentgenogram shows a definite reduction in the size of the main pulmonary artery and of its branches.

THE FATE OF OIL PARTICLES IN THE LUNG AND THEIR POSSIBLE RELATIONSHIP TO THE DEVELOPMENT OF BRONCHIOGENIC CARCINOMA*

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ST. LOUIS, MISSOURI

THE deleterious effect produced by the injection of certain oils into the lung has long been recognized; the roentgenological appearance of the pulmonary reaction is well known; certainly, at the present time, it is the exceptional case where detrimental oily material is prescribed by a physician as medication for the nasal or air passages. It is the purpose of this paper to emphasize a less obvious method by which oil may gain entrance into the lung and another, more remote, but even more serious possible consequence of its aspiration.

Guieysse-Pellissier[12] in 1920 examined the effect of injection of olive oil into the lungs of dogs and rabbits. According to his observation, droplets of oil present in the alveoli were engulfed by large mononuclear cells. The oily droplets were thus removed from the alveoli by phagocytosis without inflammation, leaving the lung structure unharmed. The olive oil used in his experimental investigation was undoubtedly highly refined and free from fatty acids. Other oils, chaulmoogra oil, olive oil, and liquid petrolatum, injected into the lungs of rabbits remained much longer in the lungs and caused "proliferative bronchopneumonia" (Corper and Freed[5]).

In 1927, Pinkerton[18] reported 6 similar cases in detail, describing the lung picture and discussing the nature of the reaction. He discussed the possibility of endogenous origin of the oily material from body fats but came to the conclusion that, "These facts together with the nature of the reaction on the part of the lung, all seem to substantiate the exogenous origin of the material much better than its endogenous origin."

It was left for Pinkerton,[19] in 1928, through experimental investigation, to determine the character of the reactions to various types of oily substances, vegetable, animal, and mineral, and to establish the criteria for an innocuous, safe oily substance as a vehicle for medication introduced into the lung.

The intratracheal injection of iodized oil (lipiodol, lipiodine or iodochloral) for bronchography is a procedure which has been rendered safe and has been carried out without hesitation by radiologists and other physicians for many years without ill effect. All of these preparations have vegetable oil bases (poppy seed oil, oil of sesame, etc.), and are free from fatty acids. Microscopic examination of the lung tissues for prolonged periods fails to show evidence of irritation or inflammation. Pinkerton's observation that these highly refined vegetable oils, free from fatty acids, do not cause inflammation reaction in the lungs, has been borne out clinically by years of use by physicians.

The opaque character of the iodized oil, however, permits observation upon the method of its entrance into the finer structures of the lung, its distribution and its final absorption and elimination. It is reasonable to suppose that the sequence of events observed with the entrance of iodized oil into the lung are duplicated in a similar fashion on the introduction of other non-opaque oils (Sante[22]). After intratracheal injection, the oil passes by gravity

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into the smaller bronchioles. At this point it is hampered in its passage into the finer lung structures by the capillary size of the tubules, the air remaining in the alveoli, and the viscosity of the oil. Immediately after introduction, the oily substance fills the bronchial tree down to the finer bronchioles, but the alveoli still remain free. After a few minutes, in the normal lung, the oil passes from the bronchioles into the alveoli leaving only remnants of the oil clinging to the walls of the bronchioles to outline their positions. The passage of the oil into the alveoli is facilitated not only by gravity and normal respiration or coughing, but also by the absorption of air from the obstructed air sacs, the oil constituting a plastic foreign body which is sucked in as the air is absorbed. The feathery appearance of the oil as seen in the alveoli is due to partial filling of the air cells; many remain entirely free from any of the oily material. This is due to the fact that the volume of the bronchioles is much less than that of the alveoli with which they communicate. If, as was frequently done in the early days, the bronchi are heavily cocainized, the cough reflex will become abolished and the bronchi will dilate. Under these conditions the bronchi become large reservoirs of oil which passes into and completely fills the alveolar structures, giving rise to the roentgenographic picture, often referred to as "drowned lung." To avoid this in our present-day examinations, only a small amount of anesthetic is sprayed on the throat or the examination is made without local anesthesia.

What is the fate of *vegetable oil* thus introduced into the lung? Within a short time any oil remaining in the bronchial branches is coughed up and expectorated, leaving only the feathery deposits of oil in the alveoli. Once foreign material has entered the air sacs it cannot be removed by coughing or ciliary action; only by the action of phagocytic cells can each small particle be carried away into the lymphatics. It may require many months for the last roentgenologic evidence of the radiopaque oil to disappear from the parenchyma.

Microscopic examination of the lung tissue reveals the oil droplets within the alveolar structures. Enormous macrophages give the appearance of signet rings as they surround and engulf the droplets of oil. By this means the oil is removed from the alveoli and carried off by the lymphatics. Autolysis of the oil is followed by complete absorption and removal. Long after all evidence of lipiodol has disappeared from the roentgenogram, oil droplets can be found on microscopic examination. In time, every vestige of oil will disappear without evidence of damage to the lung structures. Microscopically, little, if any inflammatory reaction can be seen with vegetable oils such as those used in lipiodol. This is probably due to the complete freedom of these oils from fatty acid. Deteriorated oil, therefore, should never be used because of the possibility that it might develop fatty acids which cause irritation and damage to the lung. A bottle, once opened, should never be used for subsequent examination; any change in color is likewise evidence of deterioration.

Let us consider now the effect of *animal oils*, such as cod-liver oil, upon the lung. Having demonstrated by roentgen-ray and subsequent microscopic examination, the method by which lipiodol gains entrance into and is finally eliminated from the lung, it is logical to assume that other oils may follow a similar course. Microscopic examination of the lung, after injection of cod-liver oil, would indicate that the oil finds its way to the alveoli in a manner similar to that observed with lipiodol. Once it reaches the air cells, however, it remains for a long period of time before it can be effectively removed by phagocytosis. Even when fresh, cod-liver oil contains a high percentage of fatty acids; its position in the alveoli, subject to the ebb and flow of air during respiration, favors oxidation and deterioration. The oil becomes stringy and sticky and large amounts of fatty acid are formed. As a result, the sojourn of animal oils in the lung is accompanied by

an intense inflammatory reaction and extreme fibrosis. There is marked cellular infiltration of the surrounding lung structures and large multinucleated foreign body giant cells make their appearances. A pneumonic process develops with high fever, rapid respiration and pulse with varying degrees of collapse of the patient. The quantity of animal oil, the amount of fatty acid which it contains, and the presence or absence of bacterial infection influence the severity of the reaction. Such an inflammatory process is spoken of as lipoid pneumonia. It may be fatal.

The acute pneumonic reaction following aspiration of *liquid petrolatum* into the lungs was first pointed out by Laughlen[15] in 1925. A peculiar histopathological picture was noted, the consolidation of the lung tissue being caused largely by accumulations of oil-laden phagocytes and inflammatory cells from the attendant infection.

Pinkerton[18] in 1927 reported multiple firm fibrous nodules in the lung consisting of globules of liquid petrolatum (mineral oil) imbedded in dense fibrous tissue. These tumor-like masses he called "paraffinomas."

Ikeda,[13] ten years later, pointed out that lesions resulting from aspiration of oil or fatty material into the lung fall into two

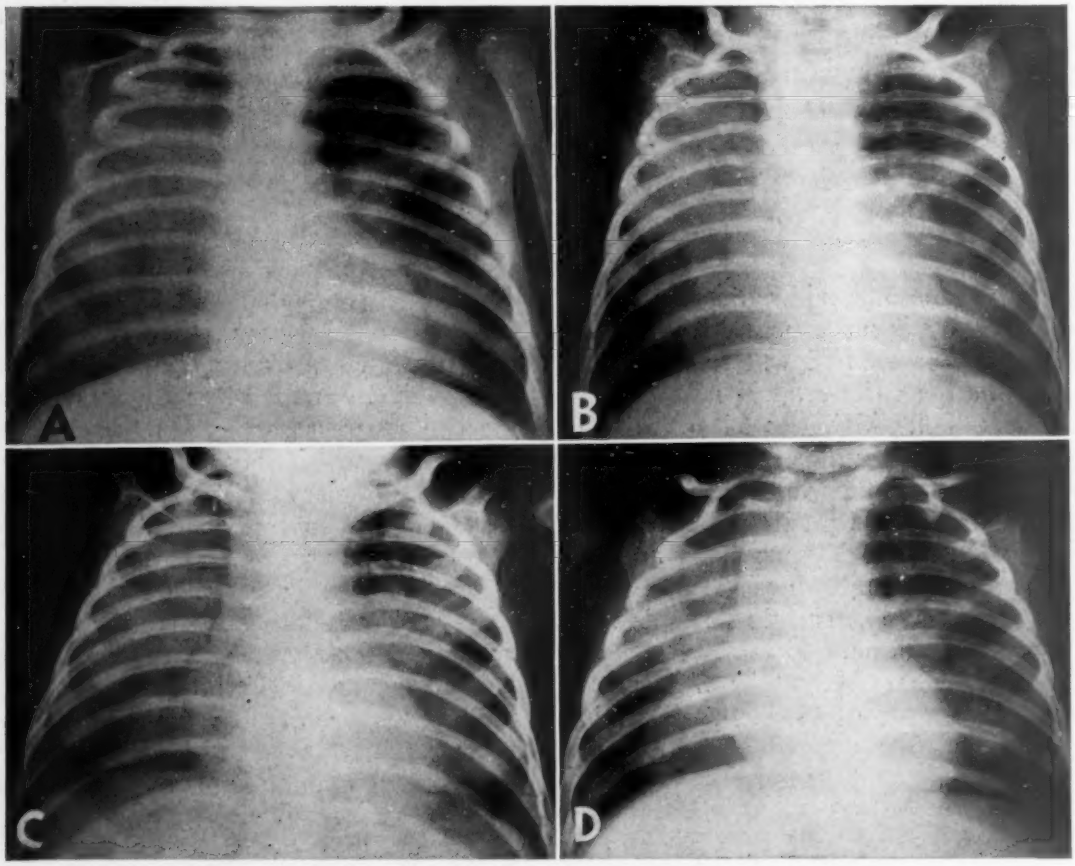

FIG. 1. Acute lipoid pneumonia, following administration of a tablespoonful of mineral oil twice daily for a two week period to an infant two weeks old. Note the diffuse homogeneous appearance of the pneumonic reaction throughout the lungs, extending to the peripheral structures. There was very little change in appearance over a period of two months (*A*, *B*, *C*, *D*) and the infant did not recover. The degree and seriousness of the reaction depends upon the amount of oil aspirated into the lung, and the rate at which it is delivered.

groups: one the *acute lipoid pneumonia* or *infantile type*, and the other the *chronic granulomatous infiltration* or the *adult type*. Since either type of lung reaction may occur in infants or adults, it would seem more logical to dispense with the age factor and designate the two types as acute or chronic with the types of lung reaction seen in each. More recent considerations of the subject would seem to bear out these observations.[6,7,14,21,23]

Mineral oil also may cause havoc in the lung, but not from the same cause, and not with the same reaction. Once in the alveoli it cannot escape except by emulsification and the action of phagocytic cells. Within the cell and in the lymphatics its behavior is different. Being inert, it cannot readily be autolyzed by the cells and it is carried with little change through the regional lymphatics to the hilum. It excites an extreme degree of cellular reaction which results in fibrosis and scar tissue which is dense and hard from contraction and becomes almost acellular. The phagocytic cells themselves may be destroyed by the indigestible oil which they contain. Such a mass of paraffin oil droplets enmeshed in heavy scar tissue gives rise to a "paraffinoma." These may attain several centimeters in diameter. They usually cluster about the large bronchial branches near the root of the lung at the site of the hilar lymph nodes—indeed, it is a question whether or not they may not even replace the lymphoid tissue of the node from pressure atrophy.

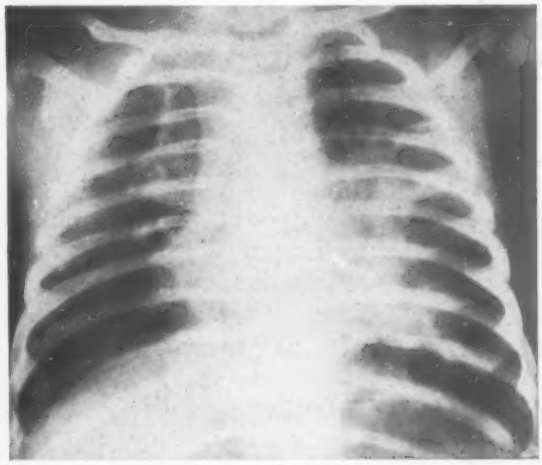

Fig. 2. Acute lipoid pneumonia, resulting from administration of a large amount of mineral oil delivered to an infant over a much longer time, many months, used in nose drops. Note the shaggy appearance of the infiltrated area within the central zone, leaving the peripheral zone clear. This indicates clearing of the alveolar structure and transportation of the oily material by the lymphatics to the hilar regions; it is a favorable sign for recovery but does not mean that the oil will be eliminated from the body.

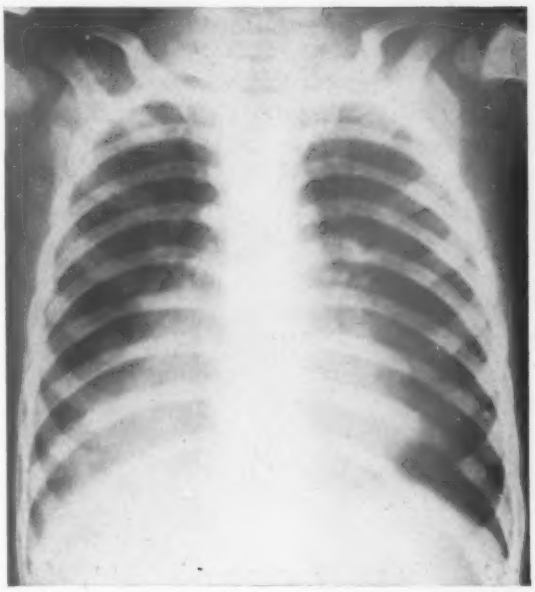

Fig. 3. Acute lipoid pneumonia in an older individual from aspiration of mineral oil taken by mouth. The acute form is the type usually seen in infants but it may occur in older children and adults also. Note pneumonic consolidation in lower portion of right lung.

Such lipoid material may pass through the hilar nodes and into the thoracic duct entering the systemic circulation (Pinkerton and Moragues,[20], Young, Applebaum and Wasserman[24]). Oil deposits have been found in the liver, spleen, and other organs of the body. Once mineral oil gains entrance into the alveoli, it is a question whether it is ever eliminated; being an inert material incapable of saponification, it resists all efforts of the phagocytes to digest it. Since it cannot be eliminated, it is evident that small amounts, if added daily, will accumu-

late to produce larger deposits in the lungs.[1,9] Numerous other cases of this sort have been reported.[2,4,8,10,11,16]

Differential diagnosis of lipoid pneumonia and pulmonary neoplasm may be difficult or impossible.[3] A method has been devised for the aspiration biopsy diagnosis of lipoid pneumonia.[17]

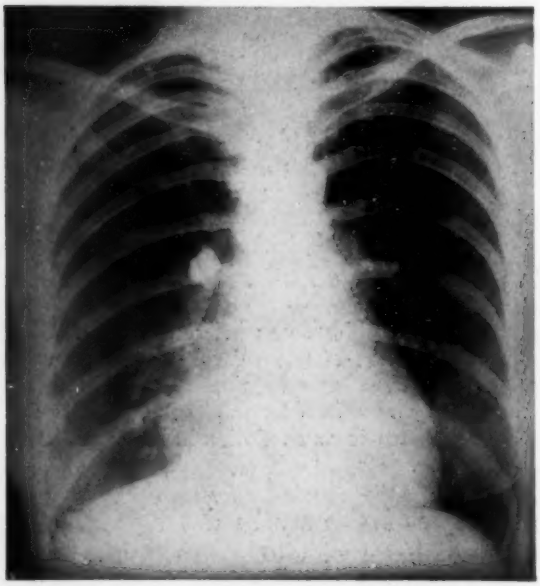

FIG. 4. Chronic type in adult showing the nodular appearance of the inflammatory process near the right hilar region, with the parenchymal portion of the lung relatively free. The patient had used mineral oil in nose drops for many years.

One would think that such paraffinomas would be innocuous once they had reached this stage, but this is far from true. They may, by pressure on larger bronchi, produce partial obstruction resulting in bronchiectatic dilatation; they may produce local inflammation and erosion, or, which seems to be of particular interest, they may be the basis for the development of bronchiogenic carcinoma. Carcinomatous tissue itself may have an appearance so grossly similar to a paraffinoma that the association of the two conditions may be entirely overlooked, unless a number of sections are taken from different areas of the tumor mass. Pinkerton,[19] in his earlier investigation (1928) had a similar impression of the possible relationship of oily substances in the lung to the development of epitheliomas as indicated by his statement, "Epithelium may participate in the repair of a suddenly produced area of necrosis in the lung, and, in so doing, may take on an almost neoplastic activity."

Our attention was drawn to the subject by the findings seen in 2 patients who died from bronchiogenic carcinoma. In one case, a large paraffinoma apparently formed the basis for the development of the carcinomatous growth. In the other case, lipoid pneumonia with early organization was present, and the association of the two lesions seemed probable. In both instances, the history of the use of mineral oil was obtained from the families after autopsy had disclosed the presence of paraffinomas of the lung.

If the microscopic appearance of paraffinoma is constantly borne in mind it may be possible that its association with the development of bronchiogenic carcinoma will prove a more frequent observation.

In ordinary microscopic preparations the fat globules appear as vacuolated areas; the fat itself is dissolved by the xylol used in preparing the specimen. In order not to lose the fat globules, all sections must be made by the frozen section method. Staining with sudan III causes the fat globules to stain bright red. If there is a question of identifying mineral oil as the fatty agent, the use of osmic acid will serve for its differentiation; osmic acid will not be influenced by mineral oil, but with all other oils it will turn black.

REPORT OF CASES

CASE 1. C. D., white male, aged sixty-two, was admitted to St. Mary's Hospital, January 7, 1947.

Family History. There was nothing of significance in the family history; no history of cancer in the family.

Past History. The patient had the usual diseases of childhood. He had never had any previous chest disease of any sort, no pneumonia, tuberculosis, bronchiectasis. No mention was made and no questions were asked concerning the use of mineral oil in any form.

Present Illness. The chief complaint on entrance was weakness, cough, mucoid expecto-

ration, and a dull pain in the lower portion of the right chest. The patient attributed his trouble to an attack of influenza which occurred about two months before entrance. Onset was with cough, fever, mucoid expectoration, and pain in the right lower chest. There was never any blood in the sputum. The fever rapidly subsided but the other symptoms continued up to the present time. The patient had lost 20 pounds in weight.

Mudd and a total pneumonectomy was performed. A diffuse infiltrating tumor mass, 10 cm. in diameter, was found in the upper portion of the right upper lobe.

Pathological Examination. The tumor was firm and the cut surface yellowish tinged with grayish-white tissue. The bronchus was thickened but not occluded.

Microscopic Examination. The architecture of the lung is destroyed and replaced in some areas

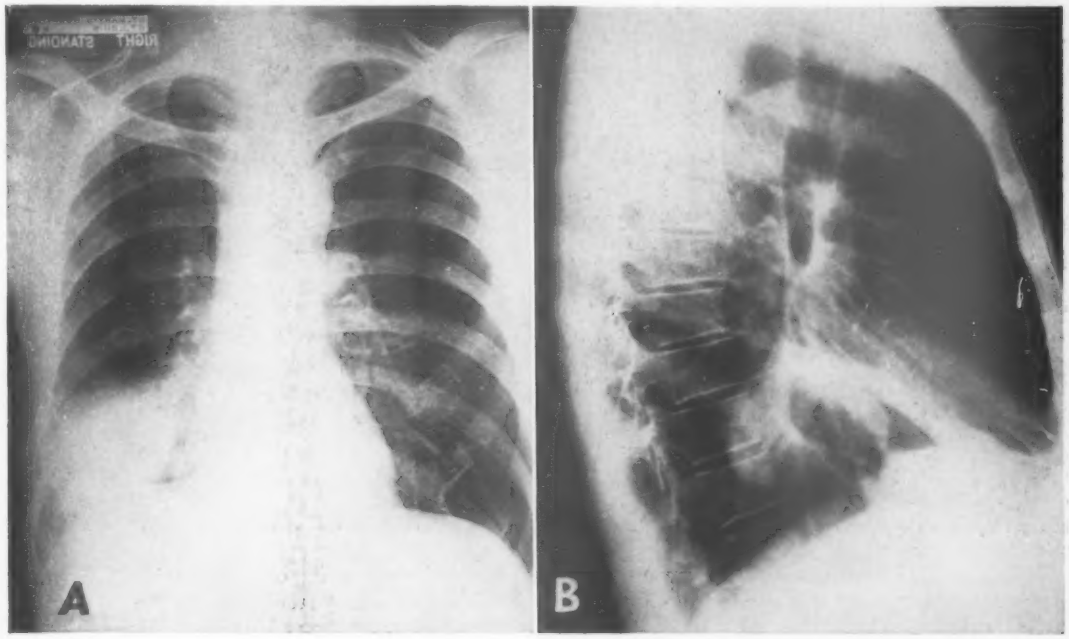

Fig. 5. Case 1. (*A*) Large rounded, well defined mass in lower portion of right lung field. (*B*) Lateral view showing location and isolated character of the mass. Correlation with the clinical history resulted in diagnosis of bronchiogenic carcinoma. Pneumonectomy specimen showed the malignant growth but association of mineral deposits in the lung. Subsequent questioning of his roommate from the institution in which he was employed disclosed the fact that he had taken mineral oil in ounce doses just before retiring for at least seven years.

Physical Examination. Physical examination disclosed impaired resonance over the lower portion of the right lung and there was some impairment on percussion but no rales. The heart was normal in rate and rhythm. There was, however, pitting edema of the lower extremities to the knees, indicating some impairment of the circulation.

Roentgen examination disclosed an oval 6 by 8 cm. mass in the right hilum region extending outward into the lung. In view of the clinical symptoms it was thought that this was due to a bronchiogenic carcinoma.

Bronchoscopy was performed but nothing abnormal could be detected.

Laboratory findings were not significant.

The patient was operated on by Dr. J. L.

by highly malignant epithelial tumor tissue. The epithelial cells are growing in sheets and cords and show marked anaplasia, pleomorphism, and hypochromatism with many atypical mitoses. In other areas the picture is that of organizing pneumonia with many vacuolated cells in the alveoli. The alveolar walls contain large vacuoles which undoubtedly represent oil droplets. In a few areas the architecture of the lung is destroyed and replaced by dense fibrotic tissue, in which oil droplets are embedded. The picture in these areas is that of an organizing lipoid pneumonia or paraffinoma. Sections from the central portion of the mass showed a combined picture of paraffinoma and carcinoma, while peripherally the picture was that of pure carcinoma. The entire picture suggests that the

carcinoma present may have arisen in a long-standing area of organized lipoid pneumonia. There is much lymphoid tissue in all sections of the lung. A section of a large bronchial lymph node shows marked hyperplasia and anthracosis but no metastatic tumor is seen. Diagnosis: (1) bronchiogenic carcinoma; (2) paraffinoma of lung.

The patient had a stormy postoperative period. Repeated transfusions were given together with stimulants, oxygen inhalation and digitalization for a failing circulation, all to no avail, and the patient died on January 24, 1947.

At autopsy there were no other significant findings.

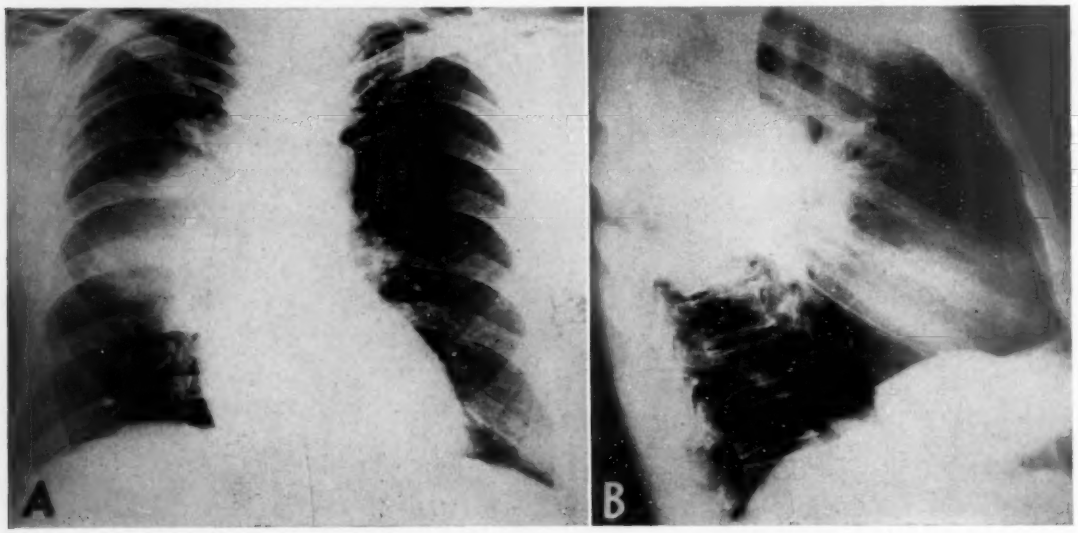

FIG. 6. Case II (*A*) Large rounded, nodular mass in right hilar region extending out into lung field. (*B*) Lateral view disclosing position and size of mass. Correlation with clinical history resulted in diagnosis of bronchiogenic carcinoma. Pneumonectomy specimen and subsequent autopsy showed large paraffinoma at hilum with superimposed bronchiogenic carcinoma. Subsequent history from his employer and relatives disclosed his use of mineral oil as a laxative in tablespoonful doses on retiring for "many years."

CASE II. M. L., white male, aged sixty-three, entered St. Mary's Hospital, St. Louis, Missouri, February 16, 1947.

Past History. On a previous hospital admission for appendectomy it had been discovered by roentgen examination that there was a mass in the right chest. There was a history of chronic cough for one year, slight expectoration but no hemoptysis, and pain in the chest. There was never any fever or signs of inflammation but merely an insidious progression of the disease and loss of 20 pounds in weight.

Physical examination was not conclusive. There was no acute distress. There was some emphysema; expansion was limited but equal. Breath sounds were accentuated in the lower lobe right posterior but no rales could be heard.

Roentgen examination revealed a mass in the right lung, which in view of the clinical symptoms it was felt was due to bronchiogenic carcinoma. The left lung was free.

None of the *laboratory findings* were of particular significance in the case; urine and sputum failed to show any positive findings; the serology was negative.

Biospy secured by *bronchoscopic examination* confirmed the diagnosis of bronchiogenic carcinoma. Following the bronchoscopic examination the patient had repeated attacks of hemoptysis for the next two weeks. He finally recovered sufficiently for operative procedure and pneumonectomy was performed.

Blood transfusions, nasal oxygen inhalation, penicillin and stimulants were given; within a few weeks the patient recovered and was discharged.

Pathological Examination. The specimen consists of the right lung having a rather firm necrotic mass in the lower lobe measuring 7 by 8 by 1 cm. in size. The necrotic area was found to be surrounded by a grayish, somewhat friable, irregular mass of tissue invading the greater

portion of the lower lobe. A lymph node was found at the hilum. Near the lower edge of the lower lobe, the lung tissue is light red in color, and a creamy yellow pus-like exudate can be expressed from the smaller bronchi. The upper and lower lobes show moderate crepitation and on section are a dull reddish brown color.

Microscopic examination. Partial destruction of the lung architecture has taken place. Masses and strands of very anaplastic, pleomorphic, hyperchromatic cells are seen having many mitotic figures; in many instances keratin pearl formation has taken place. In many areas the alveoli and alveolar walls are filled with large vacuolated mononuclear cells and many multinucleated giant cells of the foreign body type are present. Areas are present adjacent to partially necrotic regions that show spiculated structures within giant cells. Fibrosis adjacent to these areas is present but not marked. Scharlach R stains reveal that the large mononuclear cells seen in the alveolar walls and alveolar spaces are laden with oil or fat. The picture seen aside from carcinoma is that of lipoid pneumonia in the early stages of organization. Whether the carcinoma arose as a result of the lipoid pneumonia is a debatable issue in this case. The possibility that the lipoid material originated from necrotic tumor tissue cannot be excluded, but seems remote. The possibility that the picture is a reaction to lipiodol is excluded by the history. Acid-fast stains are negative. Diagnosis: (1) bronchiogenic carcinoma (squamous cell type); (2) lipoid pneumonia, with early organization.

Comment. Subsequent inquiry in both of these cases from relatives and friends disclosed that fact that they had taken mineral oil for years as a laxative, a tablespoonful or so, just before retiring. No history could be obtained of its use in any other form. The fact that small quantities of lipiodol, instilled into the nasopharynx during sleep can be demonstrated at subsequent roentgen examination in the bases of both lungs, is indication that mineral oil, especially when taken just before retiring, might gain entrance into the lungs in this way. The lack of saponification of mineral oil makes its ultimate absorption and assimilation doubtful so that oil aspiration in this manner, even if in small quantities daily, might accumulate to form a collection of sufficient size to give trouble in the lungs.

Many hydrocarbons are known to be carcinogenic agents. It behooves both radiologists and pathologists, therefore, to be constantly on the lookout for evidence of such an association. If this proves to be the case, then it is obvious that mineral oil in any form as a vehicle or for medication should be used with great caution for any purpose in the body.

SUMMARY

1. Highly refined vegetable oils, free from fatty acids such as those used in iodized oils in common use, do not produce any evidence of inflammatory reaction when injected into the lung and are ultimately completely eliminated without evidence of damage to the lung structures. When spoiled or rancid, however, even vegetable oils may become irritating and should not be used for injection into the lung.

2. Animal oils, owing to their high fatty acid content, are extremely irritating, producing lipoid pneumonia which may be fatal. If recovery occurs, however, the oily particles may be completely eliminated.

3. Mineral oil also produces damage when ingested into the lung, but this is from another cause; the oil itself cannot be saponified and therefore cannot be digested by the phagocytes, and the oily droplets thus remain. Accumulation of such droplets surrounded by dense walls of fibrous tissue are called paraffinomas.

4. Two cases of bronchiogenic carcinoma are reported, one of which undoubtedly was in pre-existing paraffinomas. In the other case, lipoid pneumonia was present, but the lesion was in an earlier stage, and its etiological relationship to the malignant lesion is less definite.

I am deeply indebted to Dr. Henry Pinkerton, Professor of Pathology and to Drs. V. Moragues and G. S. Saccomanno of the Pathology Department of St. Louis University School of Medicine, for the pathological material used in preparing this paper.

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DISCUSSION

DR. JOHN R. MCDONALD, Rochester, Minn. I have certainly enjoyed this very stimulating paper by Dr. Sante and appreciate the opportunity to discuss it.

Mineral oil granulomas are seen in many parts of the body. As far as I know, there is no evidence to support the carcinogenic properties of mineral oil. I have never seen any evidence of malignant transformation in a mineral oil granuloma.

Lipoid granulomas may be divided into three types according to the type of lipid material causing the granuloma. These lipids are the mineral oils, vegetable oils and animal oils. In my experience, lipoid pneumonitis is a very common disease. Lipoid changes can be seen in the lung in practically any chronic suppurative condition. The lipid that becomes apparent in suppurative conditions is the result of cellular breakdown in which the lipid materials, which are ordinarily not visible in the cell, become visible. This condition is seen not only in the lung but in suppurative processes throughout the body. For instance, it is very common in chronic pyonephrosis of the kidneys for the necrotic regions to be yellowish, and histopathologic examination of these regions will reveal many lipophages. As an example of an endogenous lipoid pneumonia, I should like to give data on 1 case in which a resected lower lobe of a lung revealed bronchiectasis and yellowish consolidation. Histopathologic examination revealed many lipophages, and chemical examination revealed that the lipid content of the lung was 4.1 per cent total lipid (wet weight). This was made up of 2.9 per cent cholesterol, 1.4 per cent cholesterol ester, 5.3 per cent lecithin and 1.2 per cent fatty acid. In other words, this lipid was definitely endogenous.

Furthermore, this man had no history of ever having taken nose drops.

It is often possible to find lipoid changes in the obstructive pneumonitis secondary to a carcinoma of the bronchus or any other obstruction to the bronchus. This also, in my experience, has proved to be endogenous.

I should also like to show another example of lipoid pneumonitis in which the inciting agent was iodochlorol as used in bronchography. The vehicle in this particular substance is peanut oil. In this case, the proof that this substance was iodochlorol was obtained by doing chemical analysis of the granulomatous region in comparison with a normal region of the same lung. In this particular instance the granulomatous region showed 300 times the content of iodine that the nongranulomatous region did. In my opinion, gross chemical methods are much more accurate for the determination of a specific lipid material than are histopathologic stains. Furthermore endogenous lipoid pneumonitis is much more common than exogenous lipoid pneumonitis.

A CASE OF BILATERAL PNEUMOTHORAX, ASSOCIATED WITH PNEUMOMEDIASTINUM, ATELECTASIS, PULMONARY EDEMA, AND SUBCUTANEOUS EMPHYSEMA, OCCURRING DURING LABOR: MEDIASTINAL AIR BLOCK*

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WE DESIRE to present a case of bilateral pneumothorax, associated with pneumomediastinum, atelectasis, pulmonary edema, and subcutaneous emphysema, occurring in a parturient woman immediately following delivery. The actual roentgenographic visualization of this combination of acute pulmonary accidents is of sufficient rarity to warrant mention. It is believed that the various clinical and roentgenological findings in this instance are the result, not of a fortuitiously synchronous occurrence of unrelated thoracic episodes, but of a clearly definable pattern of pathological events. This concept was originally introduced by Macklin and Macklin,[9] leading investigators of the mechanism of the appearance of atmospheric air in the interstices of the connective tissues of the lungs, pleurae, mediastinum, pericardium, and subcutaneous and retroperitoneal structures. Although the term "interstitial emphysema" is defined as the presence of air or gas in the connective tissues, the use of the phrase will be restricted, in this paper, to the occurrence of atmospheric air in the tissues mentioned.

It is felt that a review of the accumulated experimental and clinical contributions to this subject will enhance the interest which this case presents.

HISTORICAL AND EXPERIMENTAL BACKGROUND OF INTERSTITIAL EMPHYSEMA

The physiopathology of emphysema of the interstitial tissues has received the attention of clinicians since the first known recorded description of air in the soft tissues of the face, neck and chest observed in a parturient in 1783.[5] Since then, the literature is replete with reports of cases of emphysema of varying degrees of severity and occasioned by a host of causes. These reported cases are usually catalogued under the diverse headings of interstitial emphysema due to:

I. Extrinsic factors
   (1) Surgery of head, neck, chest or abdomen
   (2) Trauma to head, neck, chest or abdomen
II. Intrinsic factors
   (1) Known origin
      (a) Inflammatory or malignant diseases of the head, neck, chest or abdomen, such as influenza, malignancies of the intestinal or urinary tracts with perforation, etc.
      (b) Respiratory efforts of an unusual or violent degree occurring during illness (pertussis), the performance of physiological functions (defecation), parturition, strenuous work or sports.
      (c) Bronchial obstruction due to endogenous factors (mucus plug or proliferative lesion) or exogenous agents (toys, food, instruments).
   (2) Undetermined origin
      Spontaneous mediastinal emphysema, a syndrome introduced by Hamman[7] in 1934, and at present reported with increasing frequency.

The syndrome of benign spontaneous pneumothorax has frequently been reported in the past as an entity separate and distinct from spontaneous mediastinal

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emphysema. It is largely due to the experimental investigations of Macklin and Macklin that a unitary physiological mechanism underlying the various non-traumatic and non-surgical manifestations of atmospheric air appearing in the interstitial tissues of the lungs, mediastinum and subcutaneous tissues was conceived. According to these investigators, the basic physiologic alteration occurs in the pulmonary alveoli and, more specifically, in those alveoli whose bases abut upon bronchi, bronchioles and blood vessels in the connective tissue septa. If such alveoli are overexpanded or hyperinflated as a result of increased respiratory effort, and if the blood vessels in the supporting tissue septa cannot expand pari passu with the hyperinflated alveoli, a pressure gradient is created between the alveoli and the pulmonary vessel sheath. When the pressure gradient achieves a level great enough, rupture of the alveoli takes place with resulting entrance of minute air bubbles into the perivascular sheaths. With continued leakage of air, there is coalescence of the small air bubbles, as they are progressively propelled toward the hilum, where they may form actual blebs of appreciable size. Eventually, rupture of these blebs into the mediastinum may occur.

THE SYMPTOM COMPLEX OF MEDIASTINAL AIR BLOCK

Small amounts of air in the mediastinum may produce little or no discomfort. The presence of increasing quantities of air in the mediastinum, however, cannot long be tolerated, for there is compression of the pulmonary vessels and great vessels of the heart and mediastinum. In addition to this potentially grave threat to the mediastinal circulation, there is interference with the respiratory movements of the lungs caused by splinting action of the air in the interstitial pulmonary tissues. These two obstructive factors, the one circulatory, the other respiratory, combine to produce a symptom complex, which Macklin and Macklin have entitled "air block," and which may prove to be progressive and irreversible.

Fortunately, there are several methods or mechanisms by which this pressure may be reduced or relieved. The air in the mediastinum may dissect a pathway upward along the fascial planes toward the root of the neck, face and axillae, or downward into the retroperitoneal spaces. Again, the air from the mediastinum may rupture into the pleural cavity via a tear in the mediastinal septum, giving rise to a pneumothorax, or into the pericardium, causing a pneumopericardium.

ALVEOLAR RUPTURE AS A CAUSE OF "SPONTANEOUS PNEUMOTHORAX" AND "SPONTANEOUS PNEUMOMEDIASTINUM"

In his experiments, C. C. Macklin observed the presence of air bubbles penetrating the connective tissue of the lung from the perivascular sheaths and burrowing their way to the subpleural surface with the formation of blebs. Rupture of these blebs produced pneumothorax. More commonly, however, Macklin found that pneumothorax was due to air, accumulated under pressure in the mediastinum, erupting through the mediastinal septum into the pleural cavity. Hamman and Macklin and Macklin are agreed that the rupture of air into the pleural cavity from the mediastinum is a more logical and, experimentally, a more plausible explanation for the development of the syndrome of benign spontaneous pneumothorax than the classical theory of the rupture of a pre-existing subpleural emphysematous bleb, idiopathic or congenital.

As regards those cases of pneumomediastinum which appear to have arisen spontaneously, the Macklins believe that the modus operandi is exactly the same as in those cases in which an exciting cause is apparent or known, i.e., alveolar rupture with propulsion of coalescing air bubbles toward the mediastinum, and rupture of the mediastinal septum with production of a pneumomediastinum. In these cases of

spontaneous mediastinal emphysema, the Macklins mention the possibility of a depression of thoracic muscle tone with resulting small areas of atelectasis, which provoke a compensatory alveolar ectasia of the surrounding lung tissue. The stage is thus set for alveolar rupture. The occurrence of lesser degrees of bronchial obstruction, clinically unrecognized, but sufficient to cause alveolar overdistention, is likewise suggested. Hamman was of the opinion that alveolar walls may be stretched and remain overinflated as a result of these exciting factors, and rupture hours after the original initiating cause.

In reviewing the 39 cases of spontaneous interstitial emphysema of the mediastinum collected by Fagin and Schwab,[3] the following information relative to antecedent contributory factors is elicited. In 6 cases, a definite appreciable effort was revealed: playing golf, lifting a patient, dribbling a basketball, playing a trombone, playing volley ball, and vomiting due to alcoholism. In 4 cases, the syndrome appeared after a walk, in 2 during light manual labor, and in 6 cases, during the course of a respiratory infection. One patient developed emphysema while receiving fever therapy, 1 while in diabetic ketosis, 1 after coughing, 2 after driving and, in 16 cases, no contributory factor was discovered.

In another series of 7 cases reported by Schwartz, McIlroy and Warren,[16] the symptoms appeared immediately after playing tennis in 1 instance, after a long hike in another, after running in a third, during a walk in 2 cases, and 1 episode occurred while the patient was at rest.

Obviously, only a few individuals subjected to physical or respiratory efforts of an unusual degree subsequently present the syndrome of interstitial emphysema. Hamman and the Macklins think that there may exist a defect of the tissue quality of the alveolar structure facilitating alveolar rupture in certain individuals. It would be logical to expect the recurrence of episodes of interstitial emphysema in such cases. One of Hamman's cases developed episodes of spontaneous pneumothorax on five different occasions, subsequent to an authenticated instance of pneumomediastinum. Pinckney's case, cited by Fagin and Schwab, had three separate attacks of pneumomediastinum, as well as three episodes of spontaneous pneumothorax. In the case reported by Dassen and Fongi, likewise cited by Fagin and Schwab, the patient had five attacks of pneumomediastinum within a few weeks and a recurrence the following year. In one of the original cases presented by Fagin and Schwab, the patient gave a history of two prior episodes of the syndrome. In Case 6 reported by Schwartz, McIlroy and Warren, the patient, who was found to have a left pneumothorax as well as a pneumomediastinum on his first and only hospital admission, gave a history of similar attacks of chest pain and discomfort approximately five times a month for one year. These episodes were precipitated by running or physical training. A similar tendency toward recurrence is noted in one of the cases mentioned by Vanderlaan and Maresh,[18] as well as in the case presented by Schendstock.[15]

Finally, Dickie[2] recently published a study of 20 students who were found to have spontaneous pneumothorax, or spontaneous mediastinal emphysema, or a coexistence of the two conditions. Six students proved to have spontaneous pneumothorax without demonstrable mediastinal emphysema, 7 students gave evidence of pneumomediastinum without demonstrable pneumothorax, and 7 students presented a combination of the two conditions. It should be noted, however, that this investigator was able to demonstrate the presence of mediastinal air by roentgenoscopy or roentgenography in only 3 of the 14 cases in which a diagnosis of mediastinal emphysema was made. The diagnosis was otherwise established clinically by the presence of Hamman's sign, a crunching or crackling bruit, synchronous with the heart sound, but not associ-

ated with respiration, and best heard along the left lateral border of the sternum. In a further analysis of the 7 cases presenting both pneumomediastinum and pneumothorax, Dickie noted a verified recurrence of the mediastinal emphysema associated with pneumothorax, as well as a prior episode of contralateral pneumothorax, unaccompanied by pneumomediastinum, in 1 case. Two more patients had verified prior episodes of uncomplicated pneumothorax, and in 2 other patients, the history was strongly suggestive of prior episodes of pneumothorax and/or pneumomediastinum. In a footnote, the author mentions 7 additional cases of spontaneous pneumothorax, 6 of which were associated with mediastinal emphysema.

In recapitulation, a tabulation of the 63 cases of spontaneous pneumomediastinum reported by the authors cited above (but not including the cases described in Dickie's footnote), indicates that the roentgen examination of the thorax revealed no evidence of pneumomediastinum or pneumothorax in 19 cases, evidence of pneumothorax but no pneumomediastinum in 22 cases, evidence of pneumomediastinum but no pneumothorax in 11 cases, and evidence of both pneumothorax and pneumomediastinum in 11 cases. The presence of pneumothorax in clinically established cases of pneumomediastinum was thus roentgenologically demonstrated in approximately one-third of the cases. In 15 per cent of the cases studied, a definite tendency to recurrence of the syndrome was observed, with an occasional patient exhibiting alternating episodes of pneumomediastinum and/or pneumothorax. Lastly, those episodes which were precipitated by a known effort (running, physical training) differed in no way, obviously, from those which appeared without demonstrable cause.

It is therefore logical to assume that the syndromes of benign spontaneous pneumothorax and spontaneous pneumomediastinum result from identical physiopathological mechanisms and that there is no basis for dissociating one or the other as a separate and distinct entity. Further, any distinction between those cases precipitated by a known cause and those arising apparently spontaneously is an arbitrary one. The need for thorough roentgen examination of the thorax in these cases must be stressed.

ATELECTASIS AS A CAUSE OF ALVEOLAR ECTASIA

It was stated above that Macklin postulated the importance of atelectasis as a causative factor in producing a compensatory alveolar ectasia. Adcock[1] subsequently reported a case of a patient who developed interstitial emphysema of the lungs, mediastinum, retroperitoneal and subcutaneous tissues while receiving fever therapy. Roentgen examination revealed a small patch of atelectasis in the left lower lung field. No evidence of pneumothorax was noted, however. In 1944, Miller[11] described the case of a soldier who developed marked respiratory distress twelve hours after a tonsillectomy performed under local anesthesia. Roentgenogram of the chest showed a massive atelectasis of the right lung. Subsequent studies disclosed the presence of mediastinal and subcutaneous emphysema. In this case, again, there was no demonstrable evidence of pneumothorax. In 1945, Lavenstein[8] submitted the record of a two year old female, Negro, who was admitted for ingestion and probable aspiration of kerosene. Two days after admission, the patient suddenly became dyspneic, and upon physical examination, crepitation of the anterior chest wall was noted. Roentgen study of the chest revealed an increase in density in the right lower lobe, ascribable to consolidation or to atelectasis. Roentgenoscopic examination later in the day showed evidence of bilateral pneumothorax, mediastinal emphysema, and pneumopericardium. Further studies of the thorax confirmed the presence of bilateral pneumothorax and mediastinal emphysema. No evidence of pneumopericardium was discovered subsequent to the roentgenoscopy, however.

Lavenstein postulated the existence of a diffuse pneumonitis following the aspiration of the kerosene, and the development of focal areas of atelectasis, surrounded by zones of compensatory emphysema, with consequent alveolar distention and ultimate rupture, resulting in the introduction of air along the vascular sheaths. Rogers[14] in 1946 described a case of bilateral pulmonary infarction and bilateral pneumothorax which may be explained on a similar basis.

INTERSTITIAL EMPHYSEMA OCCURRING DURING LABOR

The occurrence of interstitial emphysema during labor, though a frequently mentioned complication, is nevertheless rare. In 1907, Kosmak, quoted by Gordon,[5] reported a total of 77 cases in the literature. Gordon surveyed the literature in 1927 and his total of 130 collected cases included 2 of his own. Nussbaum[12] reported a case in 1937 and mentioned the occurrence of 12 additional cases since Gordon's series, but his references were not detailed. In 1938, Pendred,[13] at the occasion of a medical meeting, spoke of 1 case in his experience, and he was followed by Phillips,[13] who stated that he had observed this dramatic complication of labor 5 times in his experience. Finally, McGuire and Bean[10], Greene,[6] Faust,[4] and Stanley[17] each reported a single case. Thus, in all, 135 cases have been reported and reference made in the literature to 13 additional cases since the original recorded description by Simmons[5] appeared in 1783.

In almost every instance where details were made available, the patient was a young robust primipara, whose labor had been unusually long and difficult. In an analysis of all the cases reported since the publication by Gordon, it was determined that 33 parturients developed the syndrome of interstitial emphysema well before the actual delivery (and hence prior to the possible administration of a general anesthetic agent, reference to which is missing in most instances), 20 patients developed the syndrome after delivery, and in 3 instances, the emphysema appeared at the very moment of delivery.

Roentgen examination of the thorax in these 33 cases was reported in only 11 instances. In 3, the examination was negative, and in the remainder, there was only evidence of subcutaneous emphysema. Two fatalities are recorded in the entire series. According to Gordon, 1 case was autopsied and disclosed rupture of the pulmonary alveoli beneath the pleura, but there was no evidence of emphysema of the interstitial pulmonary tissues.

CASE REPORT

The patient, M. H., a white, twenty-four year old, robust primipara, at term, was admitted June 15, 1946, at about 2:00 A.M. Her labor pains at the time of admission were described as infrequent and of short duration. Her past history as well as her obstetrical record was not noteworthy. Her last menses had occurred September 13, 1945. Urinalysis was consistently negative; blood pressure varied from 120/80 to 130/86, and pulse rate was well within normal limits. She had gained 28 pounds during pregnancy and at the time of entry into the hospital, she weighed 178 pounds.

Some short time after admission, the patient's pains became more frequent, stronger and more effective. At 7:00 A.M., an estimated five and a half hours after the onset of labor, the patient was taken into the delivery room and prepared for delivery. A general anesthesia of nitrous oxide, oxygen and ether was begun at 7:15 A.M., episiotomy performed and low outlet forceps applied. Delivery of a normal female child was effected at 7:25 A.M., and the placenta was expressed at 7:35 A.M. The condition of the patient during labor was noted as "good" on the labor room record chart, and no untoward episode was observed by the anesthetist. At 7:45 A.M., the patient reacted and promptly began to complain of shortness of breath. This difficulty increased rapidly within the next twenty minutes, during which time the patient complained of a severe pain in the right anterior chest. The pain subsequently spread to the left chest. By 8:15 A.M., the patient was markedly dyspneic and orthopneic. The administration of oxygen by nasal catheter was then begun. The pulse at this time was 168,

respirations 40 and blood pressure 170/90. Adrenalin, minims vi, and morphine sulphate, gr. $\frac{1}{4}$, were administered hypodermically.

At 9:00 A.M., the patient was examined by a medical consultant. Examination at this time revealed a markedly dyspneic, cyanotic and restless patient, whose repirations were gasping and shallow. There was puffiness of the face and neck, and distention of the neck veins. Auscultation of the chest disclosed loud coarse rhonchi and wheezes throughout both lung fields. A diagnosis of pulmonary infarction was made and 2 cc. of papaverine was given intravenously.

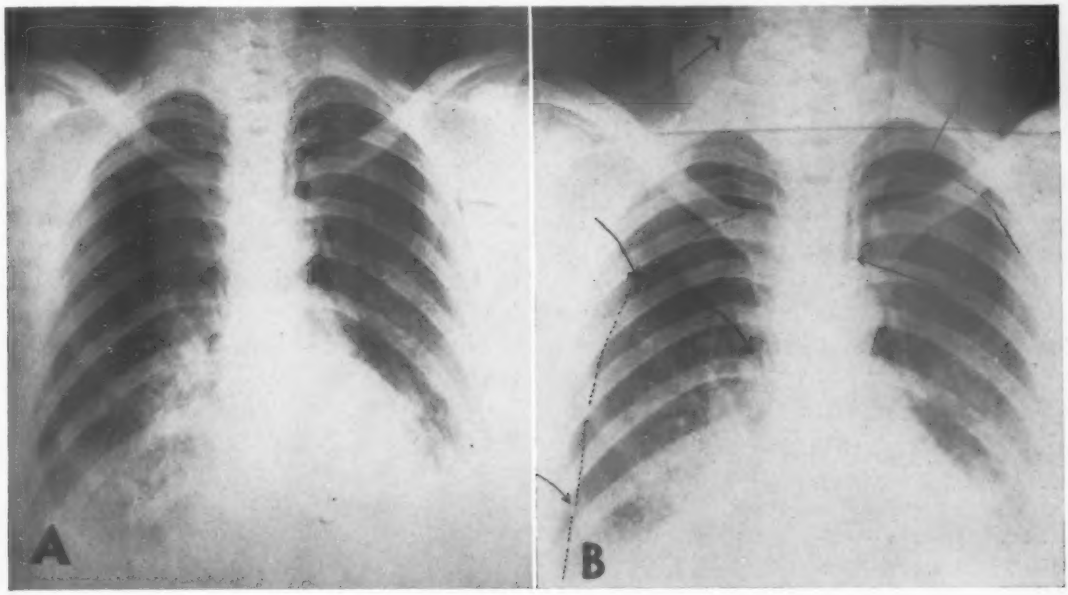

Fig. 1. (*A*) Portable roentgenogram of the chest shows the disseminated punctate densities in both lung fields, with confluence of the lesions in the right cardiophrenic angle. The subcutaneous emphysema is well visualized, but the pneumomediastinum and the bilateral pneumothoraces are difficult to reproduce (see *B*). (*B*) To facilitate orientation, dotted lines have been used to denote the location of the collapsed portions of each lung. The lower arrows point to the pneumomediastinum, the upper arrows to the cervical soft tissue emphysema.

At 10:00 A.M., a roentgenogram of the patient's chest was taken with a portable unit. This examination (Fig. 1) showed the presence of innumerable small rounded areas of density throughout both lungs. There was coalescence of these areas of density into larger, less well defined patches in the right cardiophrenic angle. A 25 per cent collapse of the right upper lobe and a smaller degree of collapse of the right middle and lower lung fields were noted. A small apical pneumothorax was also observed on the left side. Discrete but definite linear streaks of radiolucency paralleling the upper aspects of both borders of the heart were visualized. Similar though more clearly apparent streaks were outlined coursing through the superior mediastinal area into the deep soft tissues of the cervical region, bilaterally. There was little displacement of the heart and mediastinum, and the position of the diaphragmatic leaflets was not remarkable. The following impressions were recorded: bilateral, diffusely disseminated punctate areas of density, compatible with widespread focal atelectasis, aspiration pneumonitis, or pulmonary edema; atelectasis, right lower lobe; bilateral pneumothorax; pneumomediastinum; and subcutaneous emphysema.

During the next forty-eight hours, the patient's course continued stormy. Her pulse and respirations, as well as her blood pressure, remained elevated. There was no rise in temperature. A typical Hamman's sign was elicited on the second day, and crepitation was noted on palpation of the chest wall and the soft tissues of the neck. Treatment consisted of continuous administration of oxygen by nasal catheter and small doses of codeine. A second portable roent-

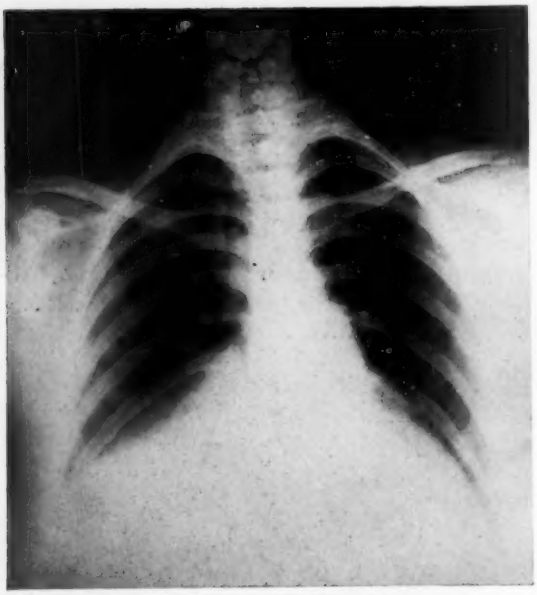

FIG. 2. Portable roentgenogram of the chest taken forty-eight hours after Figure 1. There is massive atelectasis of the right lower lobe, with displacement of the trachea and mediastinal structures to the right. The diaphragm is elevated on the right side. There is marked clearing of the previously noted bilateral, diffusely disseminated densities noted in the first examination. Residual subcutaneous emphysema is present. The residual collapse of the right lung is not demonstrated.

genogram of the chest (Fig. 2) was taken forty-eight hours after the initial study. There was noted at this time a massive collapse of the right lower lobe, with displacement of the trachea and mediastinal structures to the right, as well as slight elevation of the right leaflet of the diaphragm. There was persistence of only a small degree of the pneumothorax previously noted in the right chest, whereas the left apical collapse was no longer evident. Lastly, there was marked resolution of the diffusely scattered areas of density observed on the initial roentgenogram.

The patient improved gradually thereafter. On the tenth hospital day, the patient was permitted out of bed for short periods of time. A third roentgen study of the chest, again made at the patient's bedside (Fig. 3), showed the lung fields to be of normal aeration. The heart appeared slightly enlarged, and examination in the central department was suggested. A general physical examination, including blood pressure, pulse and temperature, as well as auscultation of the thorax, was reported within normal limits, and on the thirteenth hospital day the patient was discharged without further studies. Several months later, it was learned that the patient was again pregnant.

COMMENT

On the basis of this patient's expulsive efforts, the administered anesthesia, and the eventual clinical and roentgenological findings, the following train of events may be postulated. As the result of the increased respiratory effort incident to active labor, and the inevitable straining with closed glottis, there ensued alveolar distention. Another factor of importance in provoking alveolar distention in this case was the existence of endobronchial obstruction, probably in the form of a mucus plug, resulting in atelectasis, the presence of which is strikingly apparent in the second roentgenogram (Fig. 2). As a consequence of the compensatory alveolar dilatation, there occurred the now familiar pattern of alveolar rupture, and escape of air into the

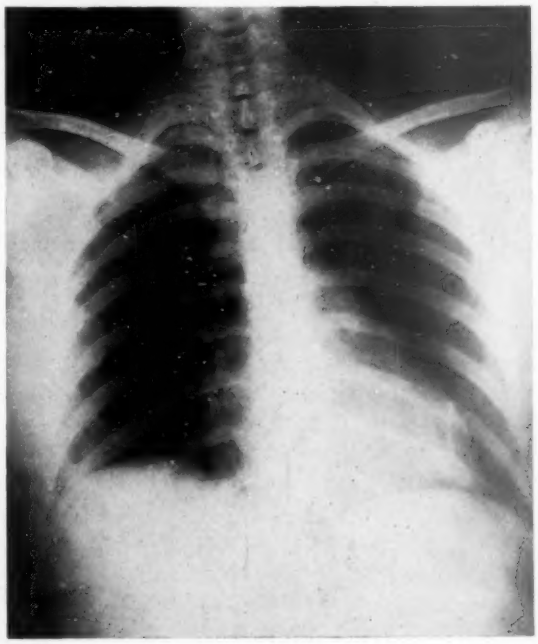

FIG. 3. Portable roentgenogram of the chest taken on the tenth hospital day. There is still residual subcutaneous emphysema noted in the left cervical region. The lung fields appear normally aerated. The heart appears slightly enlarged.

perivascular sheaths, with subsequent eruption into the mediastinum and the deep cervical soft tissues, and finally into the right pleural cavity, as was evidenced by the severe pain in the right chest. With continued leakage of air into the right pleural cavity, mounting tension developed and ultimately, there was entry of air into the left pleural cavity from a second rent in the mediastinal septum. This second pneumothorax was heralded by the spread of pain to the patient's left chest. At this moment, the patient, whose condition was then frankly alarming, presented the malignant symptom complex of "mediastinal air block."

It is of interest, at this point, to review, in retrospect, the first roentgenogram (Fig. 1). It appears very unlikely that the widespread mottled densities represent areas of aspiration pneumonitis, especially in view of the persistent lack of febrile reaction and the evanescent character of the lesions. The two other possibilities considered were focal atelectasis and pulmonary edema. The existence of massive atelectasis of the right lower lobe is not disputed (Fig. 2). However, it is less likely that the disseminated areas of opacity throughout both lungs are attributable to smaller, focal zones of atelectasis. This hypothesis could be based only upon the existence of innumerable endobronchial obstructions. Again, the lack of febrile reaction or other sequelae militate against this possibility. It would appear more reasonable to entertain the likelihood of the presence of pulmonary edema, especially in view of the symmetry and extent of involvement.

The dreaded possibility of the development of pulmonary edema as a terminal complication of "mediastinal air block" has been emphasized by Macklin and Macklin. It is felt that in the case presented there was rapid development of mediastinal embarrassment and unrelieved stasis in the pulmonary circulation with resultant edema of the lungs. The process fortunately proved to be reversible.

SUMMARY

1. The historical, experimental and clinical background of the syndrome of interstitial emphysema has been reviewed.

2. The literature of interstitial emphysema occurring in the parturient has been brought to date.

3. A very unusual case of "mediastinal air block" occurring in a parturient and affording graphic clinical and roentgenological corroboration of the conclusions empirically determined by Hamman and Macklin and Macklin has been presented.

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A STUDY OF THE EFFECT OF CERTAIN DRUGS ON CURLING OF THE ESOPHAGUS*

A PRELIMINARY REPORT

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IT IS the purpose of this paper to present a preliminary report of the effects of certain drugs on esophageal curling.

Although we have encountered 15 cases in the past eighteen months our pharmacological study was restricted to the 2 cases exhibiting severe or moderate pain.

The roentgenological incidence of curling has up to now been considered uncommon. The subject is practically unknown outside the field of roentgenology. Radiological textbooks (Templeton, Golden) mention the subject, indicating that most cases are asymptomatic. Textbooks on the gastrointestinal tract, general medicine and surgery omit mention of curling entirely.

Curling is a roentgen term describing multiple, irregular, alternating, localized contractions and dilatations of the esophagus created by incoordinated multiple segmental muscle spasms which, as pointed out by Palugyay, do not necessarily encircle the esophagus but may be located anteriorly, laterally or even posteriorly. They usually extend from the level of the aortic arch down to the level of the diaphragm and are believed by some authorities to be of myogenic origin (Golden's tertiary contractions). Most often the condition occurs in males past middle age, but occasionally it may occur during the second or third decades (Templeton). Most often curling is asymptomatic, but in 2 of our cases it produced severe to moderate pain.

During roentgenoscopy, the spasms will be seen to develop best when the esophagus is full. The contractions are more pronounced in the horizontal position. They are stationary and do not progress downward or upward. They may be evanescent or quite persistent and are independent of each other as regards extent and phase. A peristaltic wave may partially or completely eradicate the curling.

The subject has been complicated by the use of many synonyms, some of which are misleading. For example: reflex spasm (Rössle in 1912), spastic pseudodiverticula (Palugyay in 1931), segmental spasm (Bársony in 1926), pseudodiverticulosis, functional diverticula of the esophagus (Penner in 1942). All these terms presume a knowledge of the pathologic physiology. This, however, is unknown to date. In 1933 Schatzki suggested the term "Kräuselung" or curling. We favor retention of this designation until the pathogenesis is determined.

Templeton suggested that curling may be primary, unassociated with other abnormalities; or secondary to other pathological conditions. Bársony, Rössle and Teschendorf believe it to be secondary to gastrointestinal or other intra-abdominal pathological conditions creating reflex spasms in the esophagus. Penner found curling associated with laryngeal and central nervous system pathological conditions. Imbalance of the neuromuscular mechanism was suggested by Carlson. Our cases of curling were found with and without other pathological conditions.

During the past eighteen months we have studied both clinically and roentgenographically 11 cases and observed roentgenographically 4 additional cases. They were found either accidentally during preliminary observation of the esophagus prior to gastrointestinal study or in esophagrams performed for unexplained symp-

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toms. Our experience leads us to believe that the condition is not common. Undoubtedly many more cases could be found by diligent and routine esophageal studies.

Our findings support the belief of most authors that curling is due to segmental muscle spasms. Under direct roentgenoscopic observation we noted in some of the cases varying sized segments of the esophagus go into sudden spasm. The intervening portions were relaxed. The lumen may be completely or partially obliterated, creating temporary stasis in the esophagus. In other cases the curling is more or less persistent.

The fact that in 2 of our cases the severity of the pain paralleled the degree of curling prompted us to study the effects of various antispasmodics to ascertain whether or not the pain and the curling could be eliminated. In both cases other etiological factors were carefully excluded. Accordingly then, 2 cases with moderate to severe pain were selected for pharmacologic studies under roentgenoscopic and film control after:

1. Hypodermic administration of atropine sulfate gr. 1/100 every four hours for two to four doses;
2. Oral administration of tr. belladonna, 15 minims, three times a day for periods of four to fourteen days;
3. Oral administration of benzedrine sulfate, 5 mg. every four hours for thirty-six hours, and
4. Inhalation of amyl nitrite pearls.

An interval of four to seven days was allowed between the administration of the various drugs. The effects of benzedrine and amyl nitrite have never before been reported in the literature. Our findings are summarized in Table 1.

Briefly, atropine and belladonna may reduce the extent of the curling in some cases and abolish it in others. This differs with Teschendorf and Penner who reported complete disappearance of the curling. Amyl nitrite inhalation completely eliminates the curling but it recurs as soon as the effects of the drug wear off. Benzedrine also completely abolishes the curling but its effect is more lasting than from amyl nitrite inhalation.

TABLE I

PHARMACOLOGIC FINDINGS IN CURLING OF THE ESOPHAGUS

Case No.	Degree of Curling	Esophageal Pain	Associated Disease	Age Yr.	Effect of Drugs			
					Amyl Nitrite	Benzedrine	Atropine	Belladonna
I	Severe	Severe	Biliary calculus and duodenal ulcer	50	Abolished temporarily	Abolished lasting effect	Decreased	Decreased
II	Moderate	Moderate	None	52	Abolished temporarily	Abolished lasting effect	Abolished lasting effect	Abolished lasting effect

REPORT OF CASES

CASE I. C. I., white male, aged fifty, had severe esophageal pain, during meals, of fifteen years' duration, unrelieved by medications. Severe curling was demonstrated with the occurrence of esophageal pain during the segmental spasms. Amyl nitrite inhalations abolished the curling and the pain. The same effects were obtained with benzedrine. Atropine and belladonna, however, were less effective from the standpoint of elimination of pain and curling (Fig. 1 and 2).

CASE II. S. F., white male, aged fifty-two, complained of moderate esophageal pain, for many years, occurring during swallowing of food, unrelieved by medications. Roentgen studies demonstrated curling concomitant with pain. Response to amyl nitrite and benzedrine

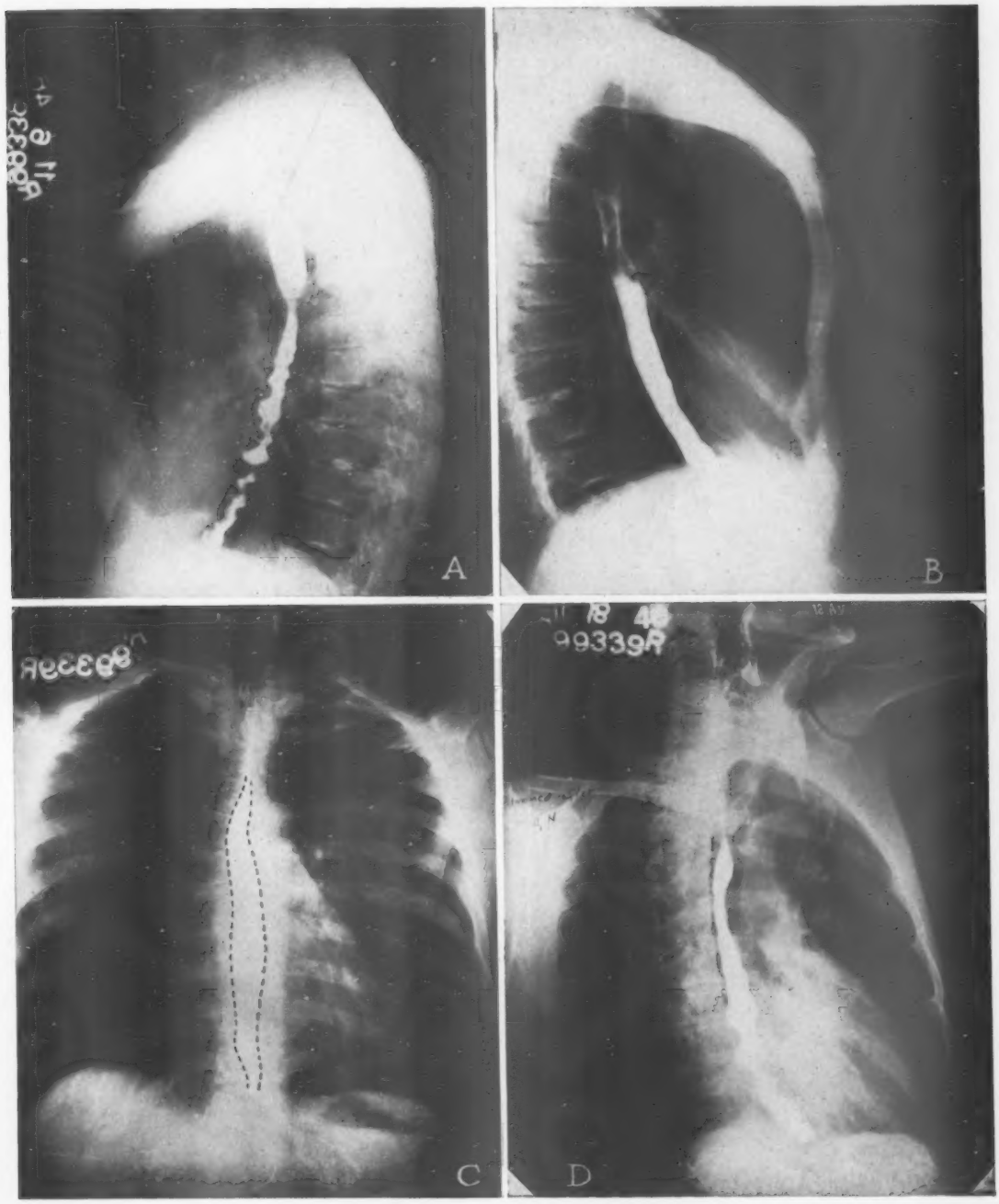

Fig. 1. Case 1. (*A*) Severe curling associated with severe pain—status just before giving amyl nitrite. (*B*) and (*C*) Total but transient relief of curling and pain during inhalation of amyl nitrite. (*D*) Return of curling immediately after amyl nitrite inhalation.

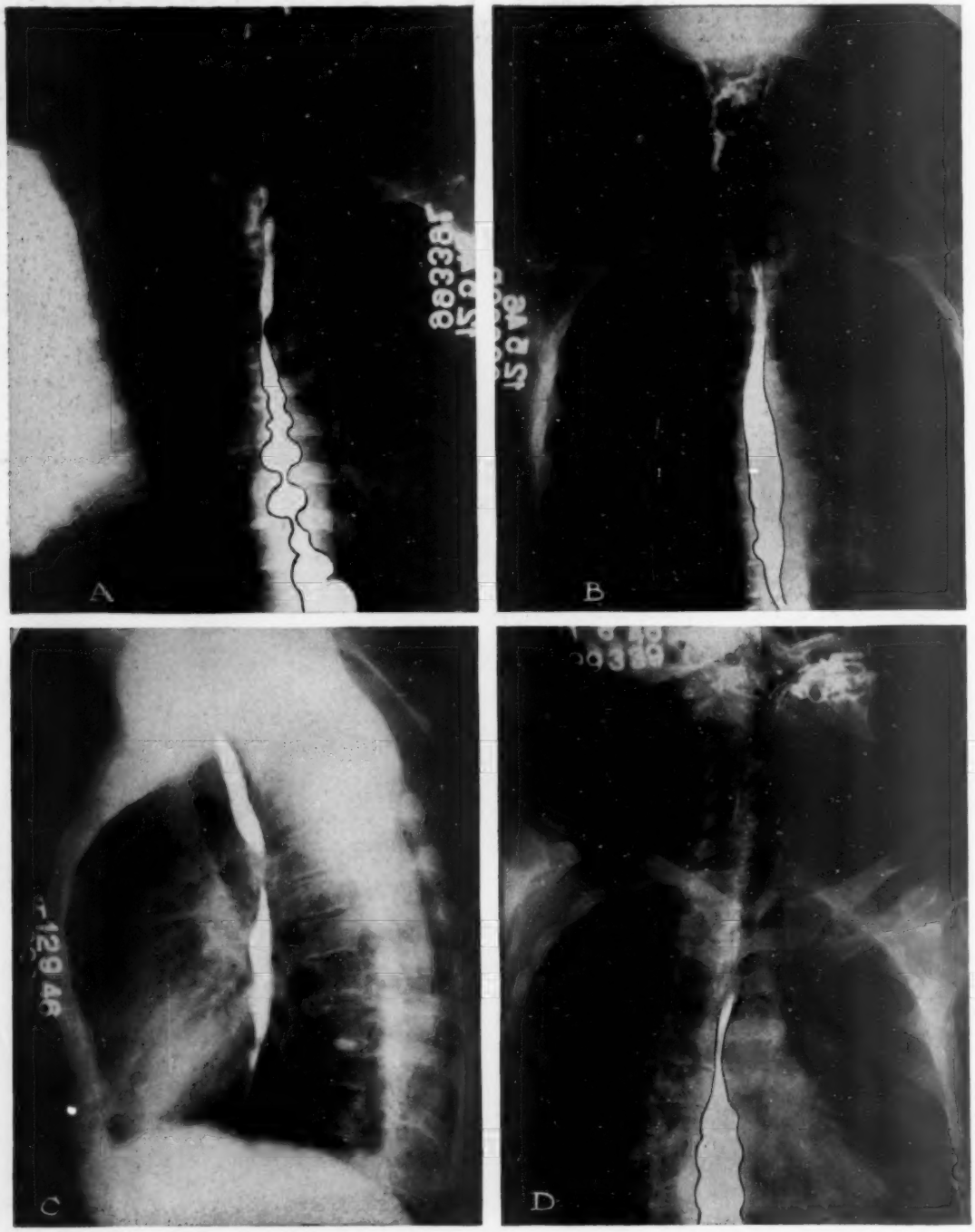

FIG. 2. Case 1. (*A*) Curling a few days before administration of benzedrine. (*B*) Total and lasting relief of curling and pain—after benzedrine 5 mg. *q4h* for thirty-six hours. (*C*) Incomplete relief of curling and pain after belladonna min. 15 t.i.d. for four days. (*D*) Incomplete relief of curling and pain after atropine gr. 1/100 (hypo) *q4h* during previous twenty-four hours.

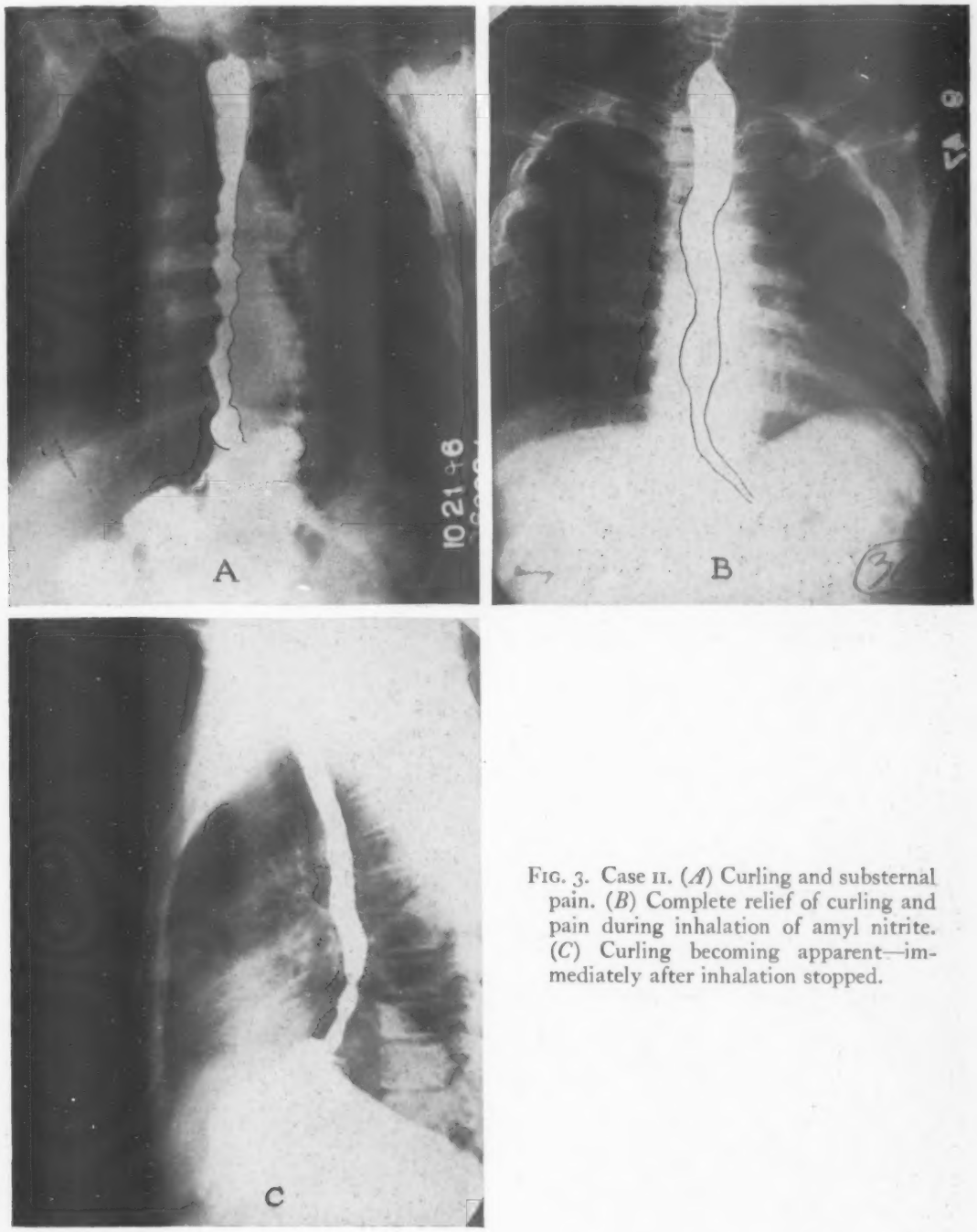

FIG. 3. Case II. (*A*) Curling and substernal pain. (*B*) Complete relief of curling and pain during inhalation of amyl nitrite. (*C*) Curling becoming apparent—immediately after inhalation stopped.

was identical with Case I. However, the patient responded just as well to atropine and belladonna with complete disappearance of curling and the pain (Fig. 3 and 4).

CONCLUSIONS

1. Curling of the esophagus is uncommon, but not rare.

2. Curling of the esophagus is believed to be produced by incoordinated unrelated segmental spasms of the smooth muscle.

3. Curling is usually asymptomatic but may produce esophageal pain.

4. Curling and its symptoms are relieved completely but temporarily by the inhalation of amyl nitrite, completely and

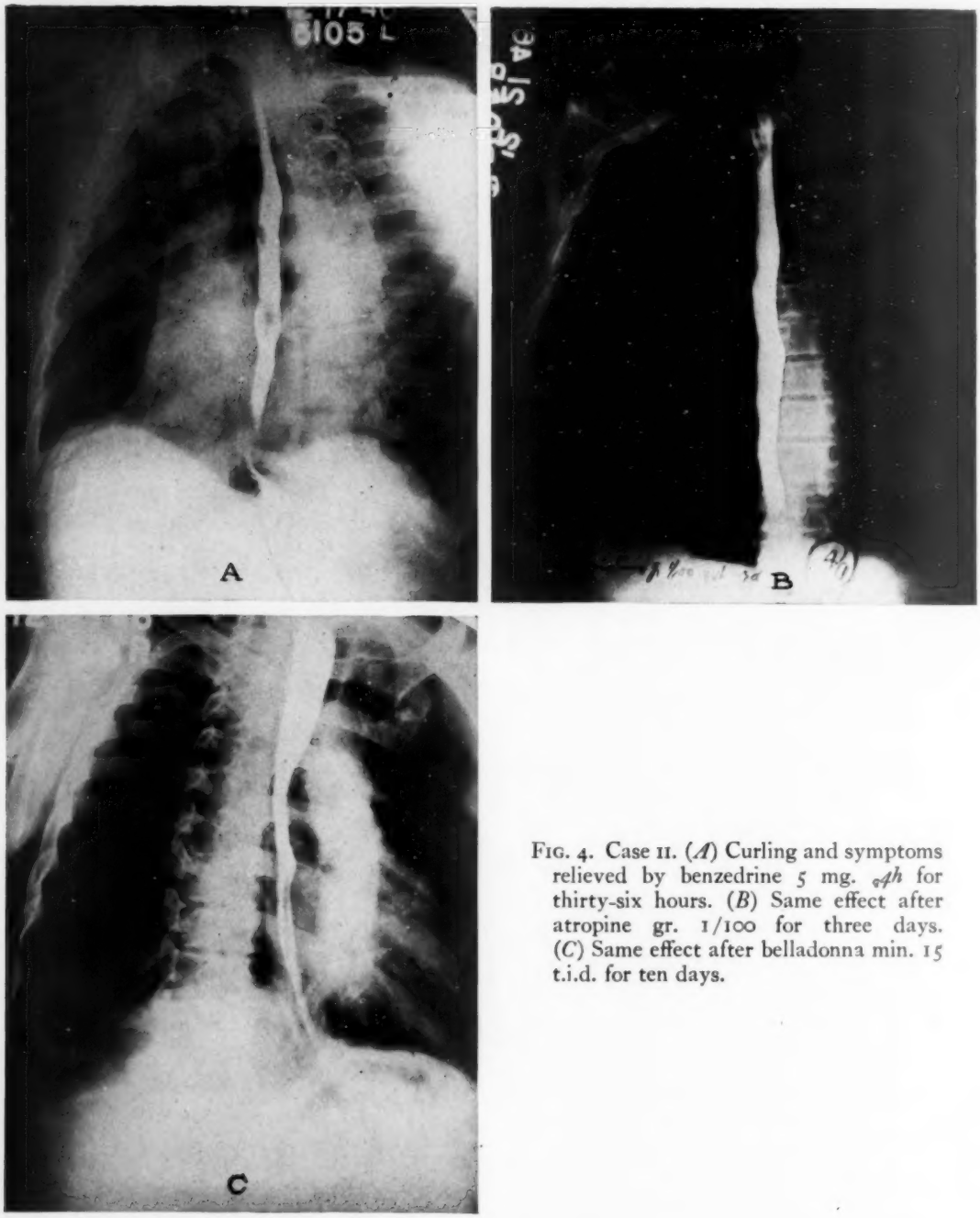

FIG. 4. Case 11. (*A*) Curling and symptoms relieved by benzedrine 5 mg. q4h for thirty-six hours. (*B*) Same effect after atropine gr. 1/100 for three days. (*C*) Same effect after belladonna min. 15 t.i.d. for ten days.

of longer duration by benzedrine, and less completely by atropine and belladonna.

5. Curling in parkinsonism is coincidental.

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EMPHYSEMATOUS CHOLECYSTITIS*

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THE roentgen demonstration of gas in the gallbladder and biliary ducts is not an uncommon occurrence at the present time. Gas may gain entrance to the gallbladder and to the biliary tree in one of three manners:

1. The most common source of gas in the gallbladder and biliary ducts is by way of a fistulous communication between the gallbladder or common bile ducts into the duodenum or colon, commonly called the internal biliary fistula.

2. Gas frequently enters the gallbladder from the duodenum through an incompetent sphincter of Oddi or following surgical procedure in which a cholecysto-enterostomy or a choledocho-enterostomy is done.

3. Gas may be found in the gallbladder and biliary ducts caused by an intrinsic infection of the gallbladder with gas-forming microorganisms. This method is the most infrequent cause of filling of the gallbladder with gas and is a rare pathological entity. The diagnosis of this condition can only be made by roentgen examination. The recognition of the process is important as a guide to therapy.

In review of the literature the authors have found that the first authenticated case in which gas was found within the gallbladder in a case of acute cholecystitis was reported by Lobingier[11] in 1908. At operation he found the gallbladder filled with gas and pus with the mucosa separated from the wall of the gallbladder containing a single stone. In 1922 Tanner[17] reported another such case and at that time he was unaware of the first report of Lobingier. Kirchmayr[9] in 1925 reported a case in the German literature. Hegner[6] reported the first case of this entity in which diagnosis was made prior to surgery by roentgen examination. In 1932 Simon[14] of Czechoslovakia reported a single case in the French literature. Wybauw[19] of Belgium, in 1936 reported a single case and in 1938 Schmidt rereported Hegner's case and added one of his own in that report. Del Campo and Otero[3] reported a case in 1940. In 1942 McCorkle and Fong[12] of California reported 3 interesting cases all diagnosed by roentgen examination. Stevenson[16] in 1944 collected 3 reportable cases all diagnosed by roentgen examination. In 1945 Pierini[13] reported a case in South America and reviewed the literature at that time. Hutchinson[7] in 1946 reported a case in England. However, his case was diagnosed at surgery and no roentgen examination was done. In the years 1927 to 1948 two cases of emphysematous cholecystitis were observed at the University of Minnesota Hospitals and two cases were found at the Ancker Hospital in St. Paul during the same interval.

GENERAL DISCUSSION

Through usage, emphysematous cholecystitis and emphysematous pericholecystitis have become the terms most commonly accepted in describing this clinical entity. Simon had employed the term pyopneumocholecystitis which may also be an adequate descriptive term for the disease. In 1922 Tanner labeled his case acute gas-producing cholecystitis, which would be similar to that of emphysematous cholecystitis. However, the term, gas gangrene of the gallbladder as used by Pierini and Hutchinson, as will be demonstrated, is a misnomer inasmuch as not all cases of gas in the gallbladder are caused by *Cl. welchii*, and not in all cases of acute cholecystitis in which *Cl. welchii* is found in pure culture is gas also found. Such cases were reported by

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Brütt,[2] Wahlberg,[18] and Koch.[10] Some authors have erroneously included these cases as emphysematous cholecystitis in their reviews.

Clinical data were reviewed in 15 of the 16 reported cases that we have found (the case of Wybauw was not included because the journal describing the case detail was not available to us). The 4 cases reported herewith are included with the 15 other cases from the literature in the analysis of the etiological and clinical data.

Etiology.

Age: The cases have ranged in age from thirty-two to seventy-four years. However, the majority of the cases are in the sixth and seventh decades of life.

Sex: There are 16 males and 3 females reported.

Bacteriology: Of the 19 cases bacteriological reports were found in 13. Of this group of 13 cases culture of the gallbladder contents from surgical specimens was made in 11. This would provide the most accurate method of determination of the microorganism present. In this group *Cl. welchii* was cultured in 7 cases, *E. coli* was found in 2, aerobic and anaerobic streptococci were found in 2 cases, and *Staphylococcus albus* was found in 2. In 2 cases of this group anaerobic cultures were not done.

In 2 of the 13 cases the cultures were made from duodenal contents obtained through a duodenal tube, and *E. coli*, *Cl. welchii* and staphylococci were found in these 2 cases. Very little significance can be given to the findings in the latter group inasmuch as these organisms are the natural inhabitants of the intestinal tract and therefore do not signify the organisms present in the gallbladder itself.

However, there is ample evidence that *Cl. welchii* is the causative organism in the majority of the cases. That the organism was found in pure culture in 7 out of 11 of the cases proved at surgery would support this contention. Gordon-Taylor and Whitby[5] were able to culture *Cl. welchii* either from the bile contents, from the gallstones, or from the gallbladder wall in approximately 10 per cent of chronically inflamed gallbladders surgically removed. Andrews and Henry[1] in a study of 91 gallbladders that were surgically removed were able to find *E. coli* present in the wall in 11 per cent of the cases and in the fluid in 16 per cent of cases and were able to culture *Cl. welchii* in the wall in 11 per cent and in the fluid in 9 per cent of the cases.

However, some of the recent authors would indicate that *Cl. welchii* is the main or possibly the only causative organism. This belief is not corroborated by a study of the facts. In 2 operated cases of emphysematous cholecystitis *Cl. welchii* was not found and *E. coli* was present. Sufficient analogous evidence is also available in the frequent occurrence of gas formation in such infections as subphrenic, intrahepatic abscesses and other abdominal abscesses caused by *E. coli*. In cases of diabetes with urinary bladder infections due to *E. coli*, the bladder is filled with gas. Gas is found around the kidney in cases of perinephric abscesses due to *E. coli* in which diabetes is present.

Association with diabetes: It was of interest to note that in the 4 cases herein reported 2 of the cases were known to have moderately severe diabetes for a number of years. Of the total of 19 cases 7 were diabetics and there was no evidence of diabetes in 4 other cases. In 8 cases of the 19 no mention was made in the report of an attempt to determine the presence or absence of diabetes inasmuch as no urinalysis or blood sugar determinations were reported.

Clinical Course. The clinical picture of emphysematous cholecystitis is indistinguishable from those cases of acute cholecystitis which are not characterized by the presence of gas in the gallbladder. The onset of symptoms is acute. Generalized abdominal pain in the epigastric region is present in most cases. The pain is later localized to the right upper quadrant with some radiation to the back and shoulders. Gradual onset of pain was noted in only a few patients. Nausea is present in most cases

and vomiting was noted in 10 of the 19 cases during the acute attack. Jaundice with the acute attack was uncommon but was found in 5 of the 19 cases during previous attacks of biliary colic. A history of previous episodes of gallbladder disease was observed in 13 of the 19 cases. Of this group the majority of the patients had an earlier history of similar symptoms and fatty food intolerance was present to a great degree. A previous history of chronic eructation, fullness and constipation was frequently encountered. No earlier symptoms were noted in 4 cases of the group and in 2 cases no mention was made in the report of any history of previous gallbladder disease.

The temperature varied from 97 to 103° F. However, most cases fell between 99 to 102° F.

Tenderness in the right upper quadrant and rigidity of the muscles and some protective spasm was present in all cases. A palpable mass in the gallbladder region was detected in 10 of the 19 patients.

The white blood cell count varies from 10,950 to 28,200 as maximum quantities. All cases in which a differential count was made exhibited neutrophilia of 80 per cent or more.

Pathology. The gross pathological appearance of the gallbladder, as demonstrated at surgery in most cases revealed a tense, distended gallbladder with thickened walls and discoloration of varying degrees. All cases contained gas within the lumen of the gallbladder with foul smelling exudate of bubbling character. In many cases the mucosa was completely separated from the muscularis and was gangrenous. Biliary calculi were found in the majority of cases. The thickened wall of the gallbladder was crepitant due to the gas within the wall and there were multiple adhesions between the omentum and the adjacent viscera, making the operative procedure even more difficult. The frequent observation of the separation of the mucosa from the muscularis affords an adequate explanation for a portion of the roentgen picture in which the gallbladder shadow is delineated by a concentric ring of lesser density representing the layer of gas between the mucosa and the muscularis.

Treatment. Of the 19 cases 12 were treated surgically. Of this group 3 died and 9 recovered. It was the impression of those authors who had seen more than one case, that if the first case was operated upon they would treat the later cases conservatively. The opinion that any form of acute cholecystitis should be treated conservatively is shared by many surgeons. Of the 3 cases that died after surgery 1 case died of a pulmonary embolus four days following surgery. Two cases died on the second postoperative day from a fulminating gas bacillus infection of peritoneum and abdominal wall. Of the 4 cases herein reported only the first case had a cholecystectomy performed. This patient recovered. The other three were treated conservatively and they recovered. Of the total group of 19 cases 7 were treated conservatively, all of whom recovered. Conservative treatment varied from practically no treatment or supportive treatments to the use of sulfonamides, antibiotics, polyvalent gas gangrene serum, and roentgen therapy in 1 case.

ROENTGEN FINDINGS

It is obvious that the clinical differentiation between cases of acute cholecystitis and those cases of acute cholecystitis in which gas has been formed by microorganisms cannot be made. The preoperative differentiation can only be made on roentgen examination. The presence of gas within the lumen of the gallbladder is probably the first stage of the roentgen appearance and this may occur any time after the onset of the initial symptoms (Fig. 2*A*). McCorkle and Fong have beautifully demonstrated cases in which serial films were made. In 1 case the first film made twenty-four hours after the onset of the symptoms showed no gas in the gallbladder and the next film twelve hours later showed gas filling the lumen of the gallbladder. If films are made in the upright position at this

stage, a fluid level may be demonstrated.

A concentric ring of lesser gaseous density surrounding the gallbladder usually involving the fundus and often extending completely around the viscus appears later (Fig. 1). Gas may still be present within the lumen. Early this shadow is uniformly demarcated. In a later stage the gas is apparently absorbed within the lumen, and the concentric, smooth, well defined ring about the gallbladder becomes streaked and bubble like in nature indicating a mixture of fluid and gas within a limiting membrane (Fig. 1, 3 and 4). The gallbladder is usually distended during the early stages, and later as one watches the absorption of the gas from the lumen the gas becomes more streaked and irregular and slowly decreases; the size of the gallbladder shadow decreases as well. The finding of these bubbles of streaked gas at an irregular distance from the wall of the gallbladder or from the air-filled lumen of the gallbladder would indicate that the pericholecystic tissues are filled with gas as well.

Feldman[4] states that a fourth diagnostic criterion for the roentgen diagnosis of emphysematous cholecystitis is the absence of gas in the biliary ducts. Del Campo and Otero,[3] however, demonstrated that gas was present in the biliary ducts as well as in the gallbladder and we have made the same observation as illustrated in Case III (Fig. 3*B*). This would indicate that the process had spread into the common duct and into the biliary ducts and a cholangitis is also present.

Many authors believe that in this disease the cystic duct is blocked by a stone. However, many cases have been reported in which no stone was found in the gallbladder and therefore this phenomenon is not necessarily a part of the picture.

We believe that the characteristic picture of streaked, "bubbly" gas within the wall of the gallbladder and in the pericholecystic tissues is pathognomonic for emphysematous cholecystitis. However, those cases in which the lumen of the viscus is filled with gas and in which no gas is present in the wall must be differentiated from an internal biliary fistula and from gas filling the gallbladder due to incompetence of the sphincter of Oddi. Kerley[8] has recently demonstrated such a case in which no such fistulous communication could be demonstrated by roentgen examination of the stomach, the duodenum and the colon. However, because of absence of constitutional symptoms in this patient he was reluctant to make a diagnosis of emphysematous cholecystitis, and at surgery a fistula large enough to pass a finger was demonstrated between the gallbladder and the transverse colon.

Fig. 1. Case I. Posteroanterior roentgenogram of the gallbladder demonstrating a concentric ring of gas surrounding the gallbladder. Note the streaked and "bubbly" character of the gas shadows. Some bubbles of gas are slightly separated from the concentric ring suggesting gas in the pericholecystic tissues.

Report of Cases

Case I. This patient, a white female, aged fifty-three, was admitted to the University of Minnesota Hospital on December 31, 1928, with complaint of attacks of right upper quadrant pain for five years. Associated with these attacks were vomiting, nausea, belching and

constipation. The pains radiated to the back and to the right shoulder. Following these attacks of pain she occasionally noticed jaundice and dark urine. Her last episode began two days before admission.

On physical examination, tenderness in the right upper quadrant with rigidity of the abdominal muscles on deep palpation was noted. No masses were felt. Temperature on admission was 99.6°F. Laboratory findings revealed a white blood cell count of 18,350 with 85 per cent polymorphonuclears. The urine was negative for sugar on two occasions.

Roentgen examination revealed the presence of multiple clustered areas of decreased density within the gallbladder and a large area of decreased density surrounding the gallbladder interpreted as accumulation of bubbles of gas

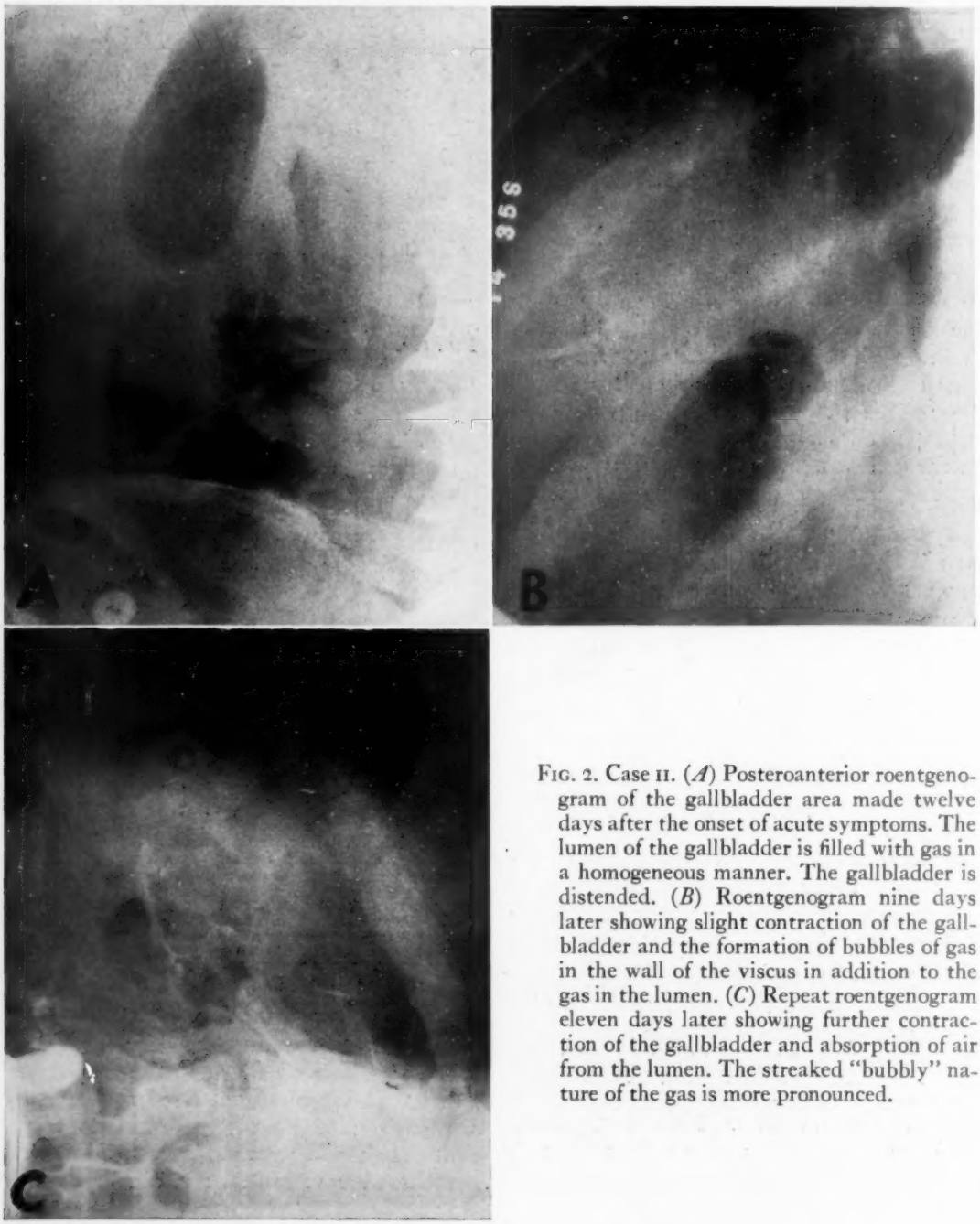

FIG. 2. Case II. (*A*) Posteroanterior roentgenogram of the gallbladder area made twelve days after the onset of acute symptoms. The lumen of the gallbladder is filled with gas in a homogeneous manner. The gallbladder is distended. (*B*) Roentgenogram nine days later showing slight contraction of the gallbladder and the formation of bubbles of gas in the wall of the viscus in addition to the gas in the lumen. (*C*) Repeat roentgenogram eleven days later showing further contraction of the gallbladder and absorption of air from the lumen. The streaked "bubbly" nature of the gas is more pronounced.

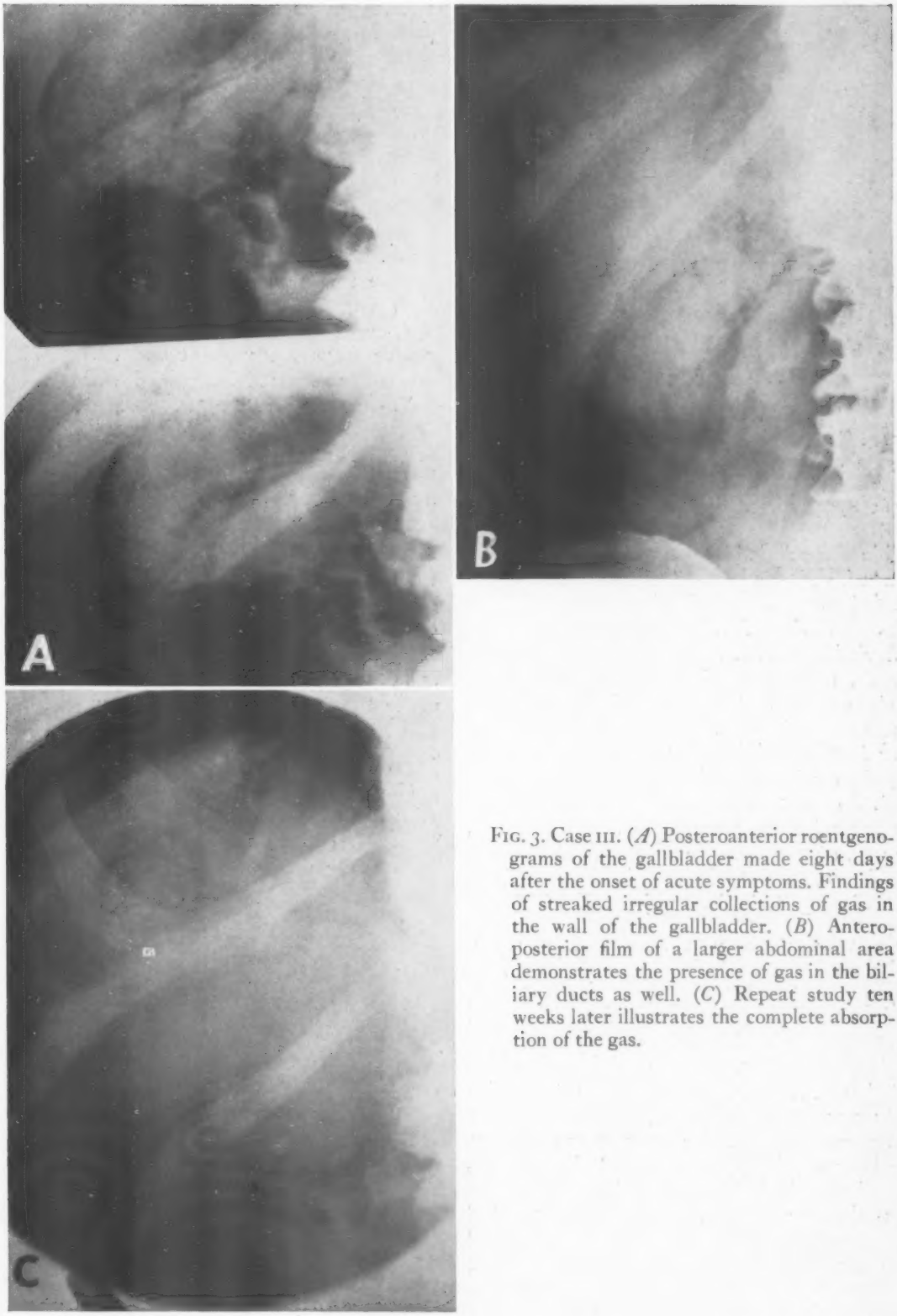

FIG. 3. Case III. (*A*) Posteroanterior roentgenograms of the gallbladder made eight days after the onset of acute symptoms. Findings of streaked irregular collections of gas in the wall of the gallbladder. (*B*) Anteroposterior film of a larger abdominal area demonstrates the presence of gas in the biliary ducts as well. (*C*) Repeat study ten weeks later illustrates the complete absorption of the gas.

FIG. 4. Case IV. Oblique posteroanterior roentgenogram of the gallbladder area exhibits mottled, streaked, bubble-like collections of gas in the wall of the gallbladder.

within the gallbladder wall (Fig. 1). Examination of the stomach, duodenum and colon showed no evidence of fistulous communication between the bowel and biliary tract.

A cholecystectomy was performed on January 10, 1929. The gallbladder was distended, thick walled and surrounded by adherent omentum and colon. The viscus was opened near the fundus and a large quantity of purulent dark colored exudate and gas was evacuated. The mucosa was entirely separated from the muscularis and contained in it a gallstone about 3 by 5 cm. Broth cultures from the gallbladder revealed staphylococci. The agar plates showed no growth. Anaerobic studies were not done.

The patient was discharged from the hospital on February 3, 1929, recovered.

CASE II. This patient, a white male, aged sixty-eight, was admitted to Ancker Hospital on March 1, 1935, with complaint of sudden attack of severe upper abdominal pain with some pressure sensation against the chest. He was nauseated but did not vomit at the onset, but later vomiting occurred and the pain became increasingly severe and localized to the right upper quadrant. This attack appeared eight days before admission to the hospital and has been constant to a great degree but had diminished somewhat in severity since the onset. He had similar attacks of right upper quadrant pain in August and later in October, 1934, but these were not as severe as the attack prior to admission.

Physical examination revealed some tenderness in the right upper quadrant and a small palpable mass was noted below the liver edge on the right. Some abdominal rigidity was also present. Physical examination was otherwise noncontributory.

Laboratory findings showed a white blood cell count of 12,950, with 86 per cent polymorphonuclears on March 8, 1935. The urine examination was essentially negative on two occasions, March 8 and April 8, 1935.

Roentgen examination on March 5, 1935, twelve days after the onset of symptoms, revealed the gallbladder filled with air (Fig. 2*A*). On March 14, 1935, a repeat examination of the gallbladder area demonstrated the gas to be of more mottled character and "bubbly" in appearance (Fig. 2*B*). On March 28, 1935, examination of the stomach was negative and the second portion of the duodenum was compressed by the gallbladder. Gas was present in the gallbladder wall on this examination but of more mottled character (Fig. 2*C*). No fistulous communication was demonstrated between the duodenum and the biliary tract. The patient's symptoms improved spontaneously with no specific therapy. Surgery was not performed and the patient was discharged on April 15, 1935, apparently well.

CASE III. A sixty-six year old male was admitted to the Ancker Hospital on December 22, 1945. The patient was a frequent visitor to the outpatient clinic and had been hospitalized frequently for arthritis, diabetes mellitus, nervousness, cardiac decompensation and pneumonia on previous occasions. Three days before admission he complained of severe pains in the epigastrium spreading to other parts of the abdomen. Vomiting of bile colored emesis occurred at the onset. Maximum pain was substernal and later was located in the right upper quadrant radiating to the back.

The essential physical findings revealed tenderness throughout the abdomen but most marked in the right upper quadrant. Some protective spasm in the right upper quadrant was noted and a mass was palpable in that area below the liver edge. The white blood count on December 22, 1945, was 28,200 with 91 per cent polymorphonuclear leukocytes. This was lower at later dates. The urine was persistently positive for sugar. The patient was a known dia-

betic for many years and had been treated at the Ancker Outpatient Clinic.

Roentgen study of the gallbladder after administration of priodax showed a normal gallbladder on September 18, 1945, but no calculi were noted. This study was done because of symptoms of fatty food intolerance. On December 27, 1945, eight days after the onset of acute symptoms, priodax was again administered and no dye was demonstrated in the gallbladder. However, the gallbladder area was completely surrounded by bubbles of gas and there was possibly some gas within the gallbladder (Fig. 3*A*). In addition, gas was demonstrated in the biliary ducts on the following day (Fig. 3*B*). A fistulous communication was not demonstrated between the colon or the duodenum. The gallbladder was examined again on March 16, 1946. There was no concentration of dye within the gallbladder but the gas shadows were no longer visible (Fig. 3*C*). The patient's acute symptoms had subsided during this interval. Surgery was not performed at that time and the patient refused elective cholecystectomy later.

Case IV. A white housewife, aged sixty-five, was well until one week before admission to the University Hospital when she suddenly developed right upper quadrant pain and nausea. This was associated with frequent belching and a sensation of gaseous distention in the midabdomen. Because of accompanying constipation she took several doses of cascara and epsom salts and this was followed on the fourth day of the illness by a severe diarrhea of frequent foul smelling liquid black stools.

Following several episodes of biliary colic, a cholecystotomy was performed in 1909 with removal of a large gallstone. An appendectomy and partial ovariectomy accompanied this operation. She weighed 95 pounds at this time and within six months she weighed well over 150 pounds and by the end of the year she became enormously obese and at present weighed over 300 pounds. Several children were born to her subsequent to the surgery in 1909. She has been a known diabetic for fourteen years using 65 units of regular insulin daily.

Physical examination revealed an enormously obese patient. Temperature was 99.6°F., pulse 92. There was a firm, rounded, tender mass palpated in the right upper quadrant underlying the scar of the previous operation. This mass appeared to be attached to the scar. It was thought possibly to be a ventral hernia.

Urinalysis showed 2 plus sugar, 2 plus acetone but no diacetic acid or albumin. The hemoglobin was 11.7, white blood cell count was 10,950, with 80 per cent neutrophils. Examination of the stool for blood with guaiac solution was negative. The fasting blood sugar was 330 The CO_2 combining power was 45 and the blood urea nitrogen 10. Liver function tests were normal, there being no increase in serum bilirubin.

Roentgen examination revealed a concentric semicircular ring of decreased density in the gallbladder area of streaked "bubbly" appearance in both posteroanterior and lateral projections. The findings were interpreted as gas in the gallbladder wall and the diagnosis of emphysematous cholecystitis was made (Fig. 4).

The patient was treated conservatively with a basic diabetic diet containing 1,130 calories and only 50 grams of fat with insulin. A course of streptomycin was given 3 grams daily for three days.

She responded to treatment, became symptom free and was discharged on October 22, 1947.

SUMMARY AND CONCLUSIONS

1. Review of the literature reveals sixteen cases of acute cholecystitis due to gas-forming microorganisms in which gas is present either in the lumen of the gallbladder, in the wall of the gallbladder or pericholecystic tissues or in the biliary ducts.

2. Four new cases of this entity are added to the literature.

3. Analysis of the significant etiological and clinical data are presented in a study of nineteen of the twenty reported cases. These cases are clinically undistinguishable from those cases of acute cholecystitis without the formation of gas.

4. The diagnosis can always be made by roentgen examination.

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LIPOMAS OF THE GASTROINTESTINAL TRACT*

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LIPOMAS are uncommon tumors of the gastrointestinal tract but are the third most common benign tumor. Adenomatous polyps are the most common such tumors and, in the experience of the Massachusetts General Hospital,[7] spindle cell tumors are more than three times as common as lipomas. Roentgen determination of the benignity of a tumor is frequently impossible. There are certain characteristics of a lipoma, however, which may aid in its diagnosis.

INCIDENCE

Only 36 cases of lipoma of the gastrointestinal tract have been found in the files and pathologic reports at the Massachusetts General Hospital since 1900. Of this group of 36 lipomas, 22 were found in 12,100 postmortem examinations, or an incidence of 0.182 per cent. This is somewhat higher than that found by Schottenfeld[13] who notes an incidence of 0.118 per cent, or 46 lipomas in a series of 37,741 autopsies collected from various sources.

TABLE I

AGE INCIDENCE OF PATIENTS WITH LIPOMAS OF GASTROINTESTINAL TRACT

Decade	Number Cases	Lipomas	
		Symptomatic	Asymptomatic*
30–39 yr.	2	2	0
40–49	6	2	4
50–59	9	5	4
60–69	15	4	11
70–79	3	0	3
80–89	1	0	1
Total	36	13	23
Average age	58.2	52.5	61.4

* Incidental finding at surgical operation or at postmortem examination.

Of the 36 lipomas, 20 were found in men and 16 in women. The average age of the patient was 58.2 years; the youngest was thirty-seven and the oldest eighty-one years (Table I). None of the lipomas was found in the esophagus; only 6 were in the stomach; 10 were located in the small intestine, and 20 in the colon (Table II). Twenty-three lipomas were incidental findings, 3 at surgical operation and 20 at postmortem examination. The remaining 13 gave rise to definite symptoms during life, although 2 of them, which were also the cause of death, were not discovered until autopsy.

TABLE II

DISTRIBUTION OF LIPOMAS IN GASTROINTESTINAL TRACT

Location	Symptomatic	Asymptomatic	Total
Stomach	0	6	6
Small intestine	2	8	10
Duodenum	0	2	
Jejunum	0	2	
Ileum	2	2	
Ileocecal valve	0	1	
Multiple	0	1	
Colon	11	9	20
Cecum and ascending colon	3	7	
Transverse colon	4	0	
Descending colon and sigmoid	4	1	
Unknown	0	1	
Total	13	23	36

The average age of the patient in whom the lipoma produced symptoms was 52.5 years. This is nine years younger than that of the group in which the lipoma was discovered incidentally, either at surgery or autopsy. Tenner[16] reported an average age of fifty-three years in a group of 12 patients who had symptoms.

* From the Department of Radiology, Massachusetts General Hospital, Boston, Massachusetts.

None of the tumors which produced symptoms occurred in the stomach; only 2 were found in the small intestine (both in the ileum), and the remaining 11 were in the colon. Those in the colon were distributed as follows: in the ascending colon 3, in the transverse colon 4, and in the descending colon 4 (see Table II). From Table III it will be noted that the symptomatic lipomas were larger, as a rule, than the asymptomatic.

Table III

Size of Lipomas of Gastrointestinal Tract

Size	Symptomatic	Asymptomatic	Total
0–1 cm.	0	7	7
1–2	0	8	8
2–3	3	7	10
3–4	1	1	2
4–5	3	0	3
Over 5 cm.	6	0	6
Total	13	23	36

The nature of the tumors is shown in Table IV. Thirty-three of the entire group were submucous, 21 being sessile and 12 pedunculated; only 3 were subserosal and these gave no symptoms. Of the lipomas producing symptoms, 54 per cent were pedunculated whereas but 25 per cent of the asymptomatic group were pedunculated. This characteristic probably contributed to the production of symptoms. Nine lipomas showing ulceration fell into the symptomatic group; in 2 other patients, no ulceration was found, although a history of blood passed by rectum was obtained. Unlike spindle cell tumors of the intestinal tract, none of this series was malignant.

Symptomatology

Symptoms had been present for periods ranging from four days to ten years. Of the 13 cases, 7 had had symptoms for less than six months, 5 for more than six months, and in 1 the duration is unknown. In a few instances, vague symptoms had been present for long periods of time, an acute exacerbation bringing the patient to the attention of the physician. Pain was experienced by 10 patients. This pain was colicky and cramp-like in nature and was usually located in the midline—epigastric, periumbilical, or hypogastric; occasionally, however, it was referred to the lower quadrants. Repeated attacks of mild pain frequently had preceded the final acute exacerbation.

Six patients complained of constipation of long duration. Anorexia, nausea and vomiting were found as a more acute symptom complex, being present from one day to three months in 5 patients. Bleeding by rectum was noted in 3 patients. One, who had had a bloody movement three days before observation, was found to have a double intussusception of the ileum induced by a lipoma; another, who had had two episodes of gross bleeding in one year unassociated with pain, was discovered to have a non-intussuscepting lipoma of the ascending colon. In the third patient who, after experiencing mild recurrent left upper quadrant pain for five years, had several "tarry" stools with vomiting for one day, a lipoma of the cecum was proved upon surgical intervention. In the last instance, intermittent intussusception had been observed roentgenographically. Abdominal distention occurred in 3 patients.

Table IV

Nature of Lipomas of Gastrointestinal Tract

	Symptomatic	Asymptomatic	Total
Submucosal	13	20	33
Sessile	6	15	
Pedunculated	7	5	
Subserosal	0	3	3
Sessile		2	
Pedunculated		1	
Ulcerated	9	0	

In 3 patients, an abdominal mass was felt. In 1, the palpable mass disappeared while the patient was under observation; he was discharged but readmitted one week

later because of recurrence. A lipoma was found in the transverse colon. Although intussusception was not present when this patient was operated upon, it would seem that the recurring mass could best be explained by such a condition. In a second patient, who complained of sharp periumbilical pain, a mass was palpated to the left and below the umbilicus; this was found at operation to be a lipoma in the ascending colon. Intussusception of the mass into the transverse or descending colon would explain its presentation as a mass in the left abdomen. The third patient had a palpable mass in the right upper quadrant, accompanied by abdominal pain, nausea and vomiting; exploratory laparotomy was negative for tumor. The patient died one month later of uremia, and postmortem examination showed a 3.5 cm. pedunculated lipoma of the ileum.

Fig. 1. Lipoma of ascending colon. Note the smooth lobulated filling defect arising from the lateral wall of the colon. This is the typical appearance of an intramural extramucosal tumor.

ROENTGEN DIAGNOSIS

As can be seen, the symptoms of lipoma are usually those of partial or complete obstruction. Often a lipoma cannot be distinguished roentgenologically from other tumors causing intestinal obstruction such as carcinoma, adenomatous polyp, spindle cell tumor, lymphosarcoma, carcinoid, and the like. In 9 cases in which roentgen examination of the colon was performed, tumor was recognized in 8; in the remaining case, obstruction at the sigmoid was observed on a plain film of the abdomen.

The roentgen signs of a nonintussuscepting lipoma are those of tumor (Fig. 1, 2 and

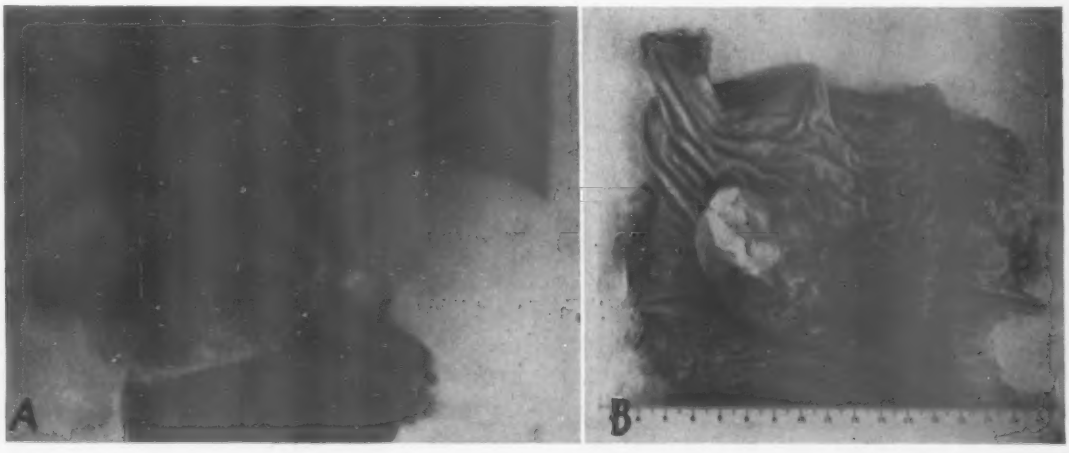

Fig. 2. Lipoma of transverse colon. (*A*) Filling defect, lobulated but smooth. Barium in folds of normal colonic mucosa surrounds the tumor. (*B*) Lipoma showing ulceration. Mucosal folds proximal to tumor are stretched. Mucosal folds distal to tumor are normal.

Fig. 3. Small asymptomatic lipoma of ascending colon found in course of investigation for diarrhea. This is perfectly smooth and has a broad base.

3). A lipoma, however, unlike carcinoma, seldom or never involves the entire circumference of the wall of the bowel. It may be sessile or pedunculated; if the latter, it may cause a dimpling or a narrowing at its point of origin. The tumor is frequently lobulated but smooth in outline; it may be ulcerated, but this ulceration may not be revealed by roentgen examination. A lipoma may produce the typical signs of an intramural extramuscular tumor as described by Schatzki and Hawes.[12] These signs are those of a smooth filling defect which, when seen in the profile view, shows an abrupt sharp angle at the point where the edge of the tumor meets the uninvolved wall of the bowel. If seen face-on, the lesion is sharply outlined in the relief picture of the bowel. The characteristic features of these lesions are obscured when the bowel is overdistended by too much barium. A lipoma showing these features may be indistinguishable from a spindle cell tumor.

In the presence of a lipoma, one significant finding occurs often enough to warrant special attention. Intussusception was evident in 6, or 45 per cent, of the 13 symptomatic cases. This percentage would rise to 62 if 2 other cases, described above, in which the combination of signs and symptoms strongly suggested intussusception, were included. In Schottenfeld's 275 collected lipomas, intussusception occurred in 36.3 per cent; Lazarus and Marks[5] report its occurrence in 45 per cent, Stetten[14] in 43 per cent, Dewis[1] in 50 per cent, and Comfort (quoted by Kirshbaum[4]) in 65 per cent.

Five of the 6 cases of intussusception in the present series occurred in the colon and were recognized roentgenographically. The sixth was a compound intussusception of the ileum and in it, the roentgenologic examination revealed a small intestinal obstruction.

Intussusception in the adult is a relatively rare condition. Eliot and Corscaden[2] state that only 10 per cent of hospital admissions for intussusception occurred in adults. Perrin and Lindsay[9] found, out of 400 patients, 18 instances of intussusception (5 per cent) occurring after fourteen years of age; in 7 of 8 cases noted in age groups twenty-nine to sixty-one, a tumor was described as causing the intussusception. Iason[3] lists the following provocative causes of intussusception in the adult: typhoid ulcers, tuberculous ulcers, dysentery, acute appendicitis, Meckel's diverticulum, a congenital ileal band, a submucous lipoma of the ileum, a foreign body, a sudden radical dietary change, and neoplasm. Tumor, however, is by far the most common cause. Schatzki[11] found a tumor to be the causative factor in all of the 11 cases of intussusception of the colon in adults seen during a period of three and a half years at the Massachusetts General Hospital.

Since intussusception so frequently accompanies lipoma, the possibility of its being caused by a lipoma should always be

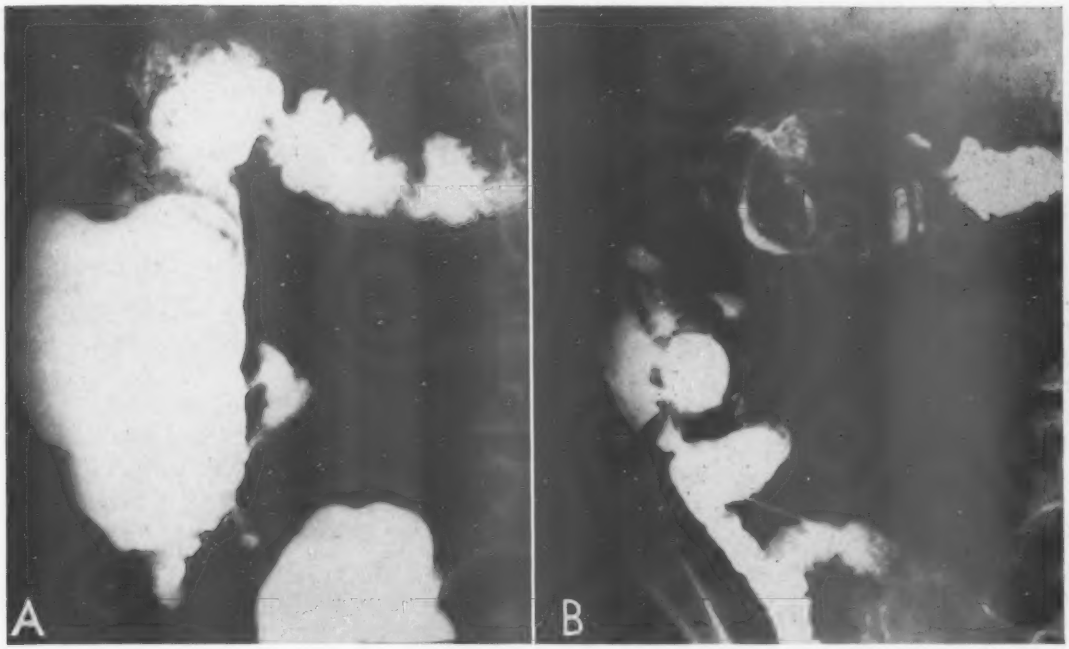

Fig. 4. Lipoma of ascending colon. (*A*) During filling of colon with barium. (*B*) Following evacuation. The tumor has intussuscepted into the transverse colon.

considered when intussusception is demonstrated roentgenologically. The signs are classical (Fig. 4, 5 and 6): the tumor usually forms the leading edge of the intussusception (called the intussusceptum, and the bowel receiving the tumor the intussuscipiens). Barium coating the folds of bowel around the tumor edge gives the characteristic appearance of multiple rings. Sussman[15] has mentioned five diagnostic criteria for intussusception: (1) obstruction to the passage of barium either by meal or by enema, but not often both; (2) a filling defect of variable size at the site of obstruction; (3) a palpable mass at the site of obstruction; (4) a change in position and shape of the filling defect following defecation; and (5) a compression of the adjacent mucosal folds, particularly well seen in the postdefecation films. Schatzki has described various roentgenologic appearances which an intussusception may assume. With an enema, the barium may stop suddenly with the formation of a cap which corresponds to the head of the intussusception. Barium may enter the sheath which

Fig. 5. Lipoma of ascending colon with intussusception. The tumor itself can be seen leading the intussusception. The barium-filled rings in the sheath are well outlined. The right abdomen, normally occupied by the ascending colon, now contains several loops of ileum.

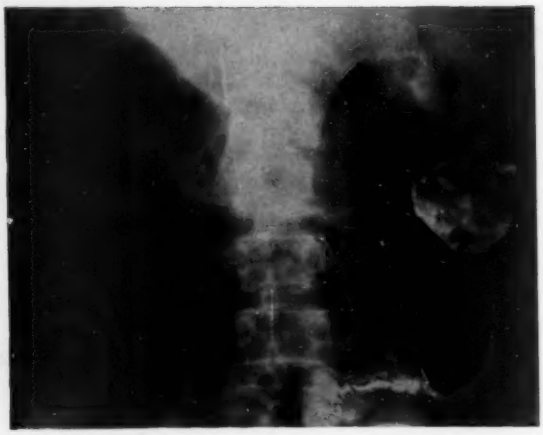

FIG. 6. Lipoma of transverse colon intussuscepted into the descending colon and causing complete obstruction.

surrounds the intussuscepted gut forming two peripheral lines of density. Frequently barium in mucosal folds forms several circular or ring-like lines surrounding the intussusception. Barium ocasionally enters the lumen of the intussuscepted bowel. This central canal is usually narrow with longitudinal rugae and is separated from the sheath by a wide, non-filled area which is occupied by two wall thicknesses of intestine and by the intussuscepted mesentery.

Examination following oral administration of barium reveals the same principal changes, except that the proximal intestine is more apt to show dilatation, particularly in cases of ileocecal intussusception. After oral administration of barium, the intussusception is usually more severe, just as it is more marked following evacuation of the barium enema. The tumor causing the intussusception, and usually forming its apex, is more likely to be outlined by barium given by enema than by mouth.

A plain film of the abdomen may be extremely useful in demonstrating an intussusception. The involved intestine is seen as a sausage-shaped, homogeneous shadow, which may be surrounded by the air-filled sheath, represented by two peripheral air stripes or air rings surrounding the "sausage." A suspicious finding is the absence of the normal feces and air-filled cecum in its usual position in the right lower quadrant, and in its place, gas-filled loops of ileum; this suggests intussusception of either the cecum or ileum into the ascending colon with dilatation of the proximal loops of ileum (Fig. 7).

TREATMENT OF INTESTINAL LIPOMAS

In the treatment of a lipoma of the gastrointestinal tract, as in all types of tumors, the importance of early diagnosis is obvious. The complications of gangrene and peritonitis can be avoided by diagnosis and early surgery. Occasionally a lipoma of the intestine is expelled spontaneously. Lazarus and Marks reported that 11 per cent of the patients in their series thus expelled the lipomas; Ratcliff[10] found 19 per cent. This is an interesting phenomenon, and it should be considered in any obscure case of intestinal obstruction in which the symptoms are relieved spontaneously.

Excision of a pedunculated lipoma or resection of the portion of the intestine containing an intussusception are the only forms of treatment. In none of the cases of this series was there any history of recurrence. The conclusion reached by Oughterson and Cheever[6] is appropriate: "Every

FIG. 7. Lipoma of ascending colon. Plain film of abdomen shows intestinal obstruction, with loops of gas-filled and dilated ileum in the right lower quadrant, and an absence of the normal feces and air-filled cecum. This suggests that the cecum has intussuscepted.

tumor of the gastrointestinal tract, especially if it is situated within the lumen of the intestine, no matter how benign the character of the growth, should be removed unless there is a definite contraindication, since it always carries with it the threat of intussusception." Another, and possibly more important reason for surgical interference is that roentgen diagnosis is still sufficiently inaccurate so that malignancy cannot be excluded.

Two cases in the present series serve to illustrate an important point in the surgical treatment of lipomas. In one case, described above, an exploratory laparotomy failed to reveal a lipoma of the ileum; in the other, roentgenologically diagnosed as a polyp of the hepatic flexure with evidence of intussusception, cholecystectomy was performed. No tumor was felt in the colon, hence it was left intact. Nine months later, because of recurring symptoms, another laparotomy was done; at this time, right colectomy revealed in the resected segment of bowel a lipoma, 9 by 6 cm., attached by means of a stalk to the wall of the colon. The tumor was described as "fluctuant and consisting of soft yellow fatty tissue." Because of this quality of softness, it has been noted repeatedly by surgeons that an intestinal lipoma, being difficult to palpate, may easily be missed even when its presence is known.

SUMMARY

Lipomas are the third most common benign tumors of the gastrointestinal tract; adenomatous polyps and spindle cell tumors are more common.

Thirty-six lipomas, seen at the Massachusetts General Hospital since 1900, have been reviewed in this report. Their distribution was as follows: stomach six, small intestine ten, colon twenty; thirty-three were submucous in position.

Thirteen of the thirty-six lipomas gave rise to symptoms; nine of the thirteen were ulcerated. Six symptomatic lipomas were causative factors in intussusception; two others probably caused intermittent intussusception. Symptoms, in the order of frequency, were pain, constipation; anorexia, nausea and vomiting; bleeding by rectum, abdominal distention, and abdominal mass.

The roentgen signs of a lipoma are those of tumor. Unlike carcinoma, lipoma seldom involves the entire circumference of the intestinal wall. It may be sessile or pedunculated; if pedunculated, it may cause dimpling or narrowing at its point of origin. A lipoma is frequently lobulated but is smooth in outline; it may be ulcerated but this may not be recognized roentgenologically. It may produce the characteristic appearance of an intramural extramucosal tumor.

Intussusception is relatively rare in the adult. It occurs, however, in association with approximately 50 per cent of symptomatic lipomas. When it is diagnosed roentgenographically, the possibility of a lipoma as the causative factor should be considered.

Any tumor within the bowel, no matter how benign its appearance, should be removed because of the possibility of intussusception and because, in most instances, it cannot be definitely differentiated from a malignant tumor. At operation, the soft consistency of a lipoma may be misleading, and detailed exploration may be necessary to locate a lipoma that has been roentgenologically demonstrated.

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DILATATION OF THE JEJUNUM

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AIR-FILLED, dilated small intestine on a plain roentgenogram of the abdomen is a well known sign of intestinal obstruction or of ileus. In addition, segmental dilatation of the small intestine, visible even without contrast media, may indicate the presence of a small intestinal tumor or inflammatory disease. Cases are presented which illustrate dilatation of the jejunum due to tumor, and due to regional enteritis.

Dilatation of a single loop of jejunum or ileum due to intestinal obstruction may last several minutes or hours, but the process is not static. If the obstruction is not relieved, other loops of dilated small intestine begin to appear and the degree of distention increases. With ileus, the entire small intestine is more likely to be air-filled and dilated, rather than a single loop. An isolated dilated loop may be caught in an internal hernial sac, in an incisional hernia, or be the site of a volvulus with multiple adhesions. Again the process is not static. The symptoms increase in severity. The caliber of the incarcerated loop increases. The wall of the dilated intestine is thin and expansile, and is not visible on the roentgenogram.

Dilatation of a single loop of jejunum may indicate tumor. The degree of dilatation is persistent and does not change from day to day. This sign is characteristic of certain non-obstructing tumors including lymphosarcoma, fibrosarcoma, highly undifferentiated carcinoma, etc. The following case is typical in symptoms, roentgen signs, and course.

CASE 1. *Lymphosarcoma of Jejunum.*

A female, aged sixty-one, was admitted to the Faulkner Hospital with a history of low grade fever and easy fatigue for one month. There were no localizing symptoms or signs. Numerous laboratory studies were inconclusive; no anemia was present. A chest roentgenogram showed no disease. A month later she was studied elsewhere for the same complaints, and no disease was found. Final admission was five months after the onset of symptoms when she returned with a low fever each afternoon, but reported the new symptoms of intermittent abdominal distention and increased flatus and belching in the last three weeks. A mild anemia was present. Tenderness was present deep in the left lower quadrant. No mass could be palpated.

Roentgen Findings. A plain roentgenogram (Fig. 1) showed a constant, stiff, air-containing loop of jejunum in the left mid abdomen. A barium swallow showed dilatation of the entire

FIG. 1. Case 1. Lymphosarcoma of jejunum. Note thickened wall.

* Radiologist, Faulkner Hospital.

FIG. 2. Case 1. Lymphosarcoma of jejunum.

duodenal loop and of the proximal first loop of jejunum (Fig. 2). Arrows distinguish the thickening of the bowel wall. The second loop of jejunum was fixed, rigid, and dilated for a distance of 8 inches. The lumen was considerably widened, and the intraluminal surface was lobulated. The barium filling made the markedly thickened wall more evident (arrows). The small bowel beyond the lesion showed the so-called "deficiency pattern" with increased segmentation, thickened transverse folds, and transient areas of distention.

At operation, an enlarged loop of jejunum with thickened, porky wall was adherent to the anterior parietal peritoneum. Its mesentery was thick, edematous, and stiff. The mesenteric changes were in the shape of a triangle with the apex at the root of the mesentery, close to the spine. A small perforation was present. The entire diseased segment and mesentery were mobilized and removed. The patient died of generalized peritoneal metastases five weeks later.

Gross Pathology. The specimen consisted of a loop of small intestine with attached mesentery. The loop was approximately 30 cm. long. Only about 5 cm. at either end had a fairly normal appearance. The entire central portion comprising 20 cm. was converted into a greatly thickened, but also dilated, mass measuring 7 cm. in diameter near its central portion where it exhibited perforations or tears on both sides, each about 2 to 3 cm. in diameter. At the site of these tears the wall was quite thin and measured 0.2 to 0.3 cm. Elsewhere it measured up to 1.0 cm. On section it had a white homogeneous, translucent surface. The lining of the distended part was somewhat granular and brownish-gray with only a suggestion of the usual mucosal folds remaining. The mesentery was converted into a dense whitish mass which on section had the appearance of enlarged, fused lymph nodes.

Microscopic Pathology. Malignant lymphoma (stem cell lymphoma differentiating into lymphoblastic lymphoma).

Comment. In a proved case of malignant lymphoma, the following roentgenological changes were observed over a period of three weeks: dilatation of a solitary loop of jejunum, thickening of the wall in the involved loop, rigidity, absence of any normal mucosal markings, broad intraluminal lobulations and altered physiological activity in the uninvolved small intestine. Lingley[11] in 1936 published 5 similar cases of "non-obstructing malignant tumors" and recognized many of these diagnostic signs. All had palpable masses, and all were rapidly fatal. Only 2 were resectable. Earlier Wakeley and Paul[16] had recognized the clincal aspects of this type of tumor, but had not distinguished this group.

The high mortality of this type of tumor in contrast to lymphosarcoma, etc., that does not exhibit these signs,[7,8,14] is probably due to the lack of obstruction. The tumor, when found, is usually of large size, with considerable infiltration of the wall and of the mesentery. The lack of obstruction and failure to become thin and to perforate leads to absence of symptoms for several weeks. A continuous low-grade fever in our case was indicative of the presence of disease for a period of four months before abdominal symptoms helped in localizing the process to the intestine. When a small intestinal tumor presents the above enumerated roentgenological signs, a grave

prognosis even before operation is probably indicated.

Dilatation of a single loop of jejunum may also be due to inflammatory disease and again is static and persistent in contrast to the dilatation of obstruction.

Case 11. *Localized Subacute Jejunitis.*

A male, aged fifty, was admitted to the Faulkner Hospital after five days of nausea, vomiting, and epigastric pain. The pain became steady and had localized itself to the left of the umbilicus. Ten years previously a gastrointestinal series had been negative.

Examination showed tenderness and spasm to the left of the umbilicus and in the left flank, a temperature of 100°F., and a leukocyte count of 12,700 per cubic millimeter. A clinical diagnosis of diverticulitis was made.

On the fifteenth day of illness, a plain roentgenogram of the abdomen showed a dilated loop of small intestine with air trapped in it. The loop had a thickened wall. The trapped air had a granular appearance. At the distal end of the lesion, two small calcified areas were seen in the soft tissues and were interpreted as calcified mesenteric glands. A barium enema showed considerable spasm of the descending colon lateral to the involved loop and no diverticula.

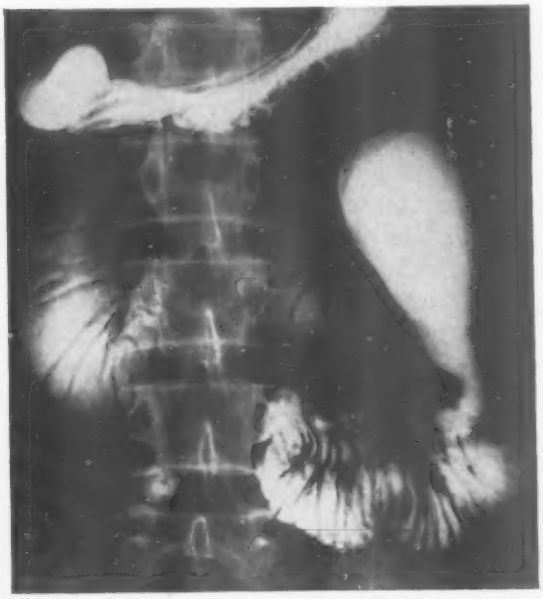

Fig. 3. Case 11. Regional jejunitis. Note dilated proximal small intestine and rigid outline of involved area.

Fig. 4. Case 11. Regional jejunitis. Note thickened wall and reticular mucosal pattern.

Gastrointestinal series proved the rigid loop to be very high in the jejunum and only about 10 inches from the ligament of Treitz (Fig 3). The duodenum and jejunum proximal to the lesion were dilated but possessed a normal, though distended, mucosal pattern. Barium rushed by rigid walls of the lesion as through a stiff tube. The lumen was dilated. The normal mucosa was replaced by a coarsely granular or lace-like reticular pattern (Fig 4). The wall was 2–4 mm. in thickness. Beyond the lesion the small intestine was very motile and exhibited unusual segmentation; in places, some of the loops were temporarily dilated. There was no definite obstruction in the dilated loops proximal to the lesion, and peristaltic waves could be seen in the wall.

In the hospital the vomiting disappeared

along with the tenderness. After two weeks, tenderness had decreased sufficiently so that a rigid, L-shaped mass could be palpated.

He was discharged home and did well for about ten days. The anorexia began associated with colicky pain in the left upper quadrant. Vomiting recurred, and he was re-admitted and prepared for operation. Roentgenograms showed the persistently dilated rigid loop, which was palpable and not tender. There was no anemia. At laparotomy, multiple adhesions were found in the left side of the abdomen. Upon freeing them, a very friable, soft, thick, purplish red loop of proximal jejunum was exposed and removed with difficulty. The mesentery feeding this loop was thickened and edematous, and the same change was seen in the mesentery to the proximal jejunum as far as the ligament of Treitz. The jejunum proximal to the lesion was distended but soft and had a normal serosa. No other diseased segments were seen in the small or large intestine. The loop was completely removed and anastomosis accomplished.

Gross Pathology. A. The specimen (representing the involved portion of jejunum) consisted of a ragged, irregular sheet of dark red tissue. Part of it measured 11 by 3 cm. and about 0.2 cm. thick; this suggested part of the small intestine. Attached to it was a sheet of similar tissue, apparently a torn portion of intestine, measuring 9 by 3 cm. What appeared to be the mucosal surface of this specimen was dusky red and granular, with none of the usual mucosal folds. The serosal surface was likewise dull red and granular. As noted above, the wall was thinner than that of normal jejunum. *B.* Also received, a segment of jejunum 13 cm. long and about 3.5 cm. in diameter with a considerable piece of mesentery attached. Except for some roughening and reddening of the serosa on one side over the attachment of the mesentery to the intestine, the surface appeared smooth and glistening. On section the mucosa showed the usual folds and appeared intact. In the mesentery were two very hard calcified nodules, each about 1 cm. in diameter and another firm whitish one about the same size.

Microscopic Pathology. A. All coats, but especially the mucosa and submucosa, were infiltrated by lymphocytes, eosinophils and plasma cells. The mucosa was atrophic and in part ulcerated. Early fibrosis was noted in the submucosa. No tubercles or parasites were seen. *B.* This part of the intestine appeared essentially negative except for eosinophils in the mucosa. A lymph node showed some fibrosis of capsule and proliferation of sinusoidal lining cells. The calcified nodules showed amorphous calcific material in lymph nodes.

Diagnosis: *Subacute jejunitis*, nonspecific, with ulceration of mucosa; chronic lymphadenitis; healed calcific lymphadenitis, probably tuberculous.

Comment. Microscopically the findings were those of a nonspecific subacute jejunitis compatible with an early stage of so-called "regional enteritis." The calcified areas were small discrete nodes in the mesentery. Regional ileitis or enteritis involving the jejunum alone is rare: Out of 507 cases collected by Ravdin,[13] 10, or 3.2 per cent, were restricted to the jejunum. The above reported case is unique and is being reported elsewhere in detail.[3]

In a proved case of regional enteritis in the jejunum, the following roentgenological changes were observed over a period of weeks: persistent dilatation of a solitary loop, thickening of the wall in the involved loop, rigidity, coarse granular intraluminal surface, distention with peristalsis of the small bowel proximal to the lesion, and hypermotility and increased segmentation of the small bowel distal to the lesion.

In tumor (Case 1)[11] and in infection (Case 11), some of the roentgenological signs in the jejunum were similar. In both, the plain roentgenograms showed a distended, air-containing, elongated cavity with a thickened wall around it. The thickening was several millimeters and was due to cellular infiltration of the bowel wall.[4,5,11,15] In both no peristalsis could be seen in the lesion, and barium rushed through it. There was no mechanical obstruction to the passage of barium through the entire small intestine.

In both diseases, the physiological activity of the adjacent small intestine was greatly altered. Peristalsis was increased throughout the small intestine in the case of tumor, and distal to the regional jejunitis. There was increased segmentation of

the barium, and transient areas of distention on serial roentgenograms. In some areas, the transverse markings were increased in width and "edematous." In Case 1, the "deficiency pattern" was simulated, though the patient was in excellent nutrition, eating well and had no anemia. It is apparent that the physiology of the entire small intestine, as exhibited with a barium medium, is altered by intrinsic disease located in a small segment of it.

A second change in small intestinal physiology was seen in Case 11 proximal to the non-obstructing lesion. Both the duodenum and the first 12 inches of jejunum were dilated, though exhibiting good peristalsis. A normal though distended mucosal pattern was clearly seen (Fig. 3). At operation the mesentery to the proximal jejunum was edematous, stiff, and porky. The intestine supplied by this mesentery was found to be distended on roentgen examination and at operation, but later was proved to possess a normal serosa and mucosa. This type of distention is to be distinguished from that due to obstruction and is probably due to disease in the mesentery itself. Severe dilatation of the small intestine is often seen in mesenteric artery thrombosis. Golden[9] reported distention of the proximal jejunum in a case of regional enteritis of the lower jejunum in which there was no mechanical obstruction, and commented "that the intestine may be dilated without ileus proximal to a mechanical obstruction." The condition of the mesentery was not proved, however.

Golden distinguished a third change in small intestinal activity when only a small segment was involved in disease. A case with carcinoma of the terminal ileum showed constriction without complete occlusion of the lumen. Several dilated loops of small bowel had been previously shown by roentgen examination proximal to the lesion. He thought the change might be physiological. In summary, the motor activity and tone of the small intestine may be greatly altered by the presence of a small area of carcinoma or inflammatory disease without obstruction.

Fig. 5. Recurrent regional ileitis. Lesion extends back from ileotransverse colostomy about 24 inches.

Differentiation between non-obstructing tumor[4,7,14,15] and inflammatory disease[1,2,6,13] may be accomplished before operation by a study of the intraluminal surface without and with barium. In obstruction and ileus, some of the normal mucosal markings are still visible though widely separated. In both tumor and infection, no normal mucosa is seen. A case with tumor may show a smoothed out, gently lobulated, and even slightly polypoid intraluminal surface. In a case of inflammatory disease, the intraluminal surface may be coarsely granular, lace-like or reticular. The pattern in the jejunum in our case was similar to that seen in typical terminal ileitis. Figure 5 shows the intraluminal pattern of a recurrent area of regional ileitis in the mid ileum. The distal 2 feet of terminal ileum had been resected two years previously. The recurrence was retrograde from an ileotransverse colostomy. The similarity between the intraluminal surface in Figures 4 and 5 is striking.

SUMMARY

In addition to early intestinal obstruction and ileus, dilatation of a loop of jejunum may occur with certain non-obstruct-

ing tumors and with certain infections. Associated with the latter two diseases, the roentgenological signs are rigidity, thickening of the wall, loss of normal mucosal markings, and alteration in the motor activity of the entire small bowel. Differentiation between tumor and infection may be accomplished before operation by study of the intraluminal surface of the diseased loop with and without barium.

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ROENTGEN DEMONSTRATION OF FETAL DEATH (GAS WITHIN THE FETUS) AND UTERINE RUPTURE

A REPORT OF THREE UNUSUAL CASES

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WITHIN the past two years 2 obstetrical cases, very similar, have been seen, both showing evidence of gas in the fetal circulatory system. One of these cases was associated with uterine rupture. The third case demonstrates both uterine and fetal (intrauterine) gas, marked in degree, due to intrauterine infection. This latter case is included because it is unusual to have the opportunity to demonstrate such changes roentgenographically.

REPORT OF CASES

CASE I. M. K., aged thirty-eight, para 2-0-0-2, was admitted to the hospital on February 9, 1945, after having been in labor for four days without progress. The patient was referred from another, out-of-town, hospital. The past and family histories were negative. The past obstetrical history revealed two normal, spontaneous deliveries, the last occurring ten years previously. The weight of the last baby was approximately 8½ pounds.

During the present prenatal course the patient was seen for the first time when she was six months pregnant. Her blood pressure was 170/110. She was placed on a low protein and low salt diet. Following this a trace of albumin was noted in the urine on several occasions. Her expected date of confinement was January 20, 1945. She entered the out-of-town hospital on February 5, 1945, because her membranes had ruptured. Since labor did not ensue she was discharged the following day with instructions to return when labor started. She was readmitted to the same hospital Thursday morning February 6, 1945, in early labor, which progressed normally throughout the day. At 11 P.M. her doctor was called and told that she was fully dilated, the presenting part almost on the perineum, and that she could be delivered. Upon examination of the patient, the cervix was found fully dilated, but the presenting fetal part was only 1 cm. below the spines and no attempt at delivery was made.

A consultant was called and he advised pantopon gr. ⅓ so that the patient would rest throughout the night. A medical induction was started Friday morning, February 7. The patient's temperature at this time was 100°; pulse 120 and blood pressure 180/110. The urine showed a 1 plus albumin. The patient rested throughout the night. The medical induction consisted of castor oil, enema, and pitocin but proved ineffectual. On February 8 a roentgen examination of the abdomen revealed the fetus in right occipital anterior position with the head markedly flexed, giving the impression of a neck presentation. A catheterized specimen of urine on Saturday showed gross blood and a 4 plus albumin. The blood pressure was 170/110 at this time. The consultant was again called and he advised another medical induction to be started Sunday morning, February 9. This was given without any change noted, consequently a second consultant was called. The latter consultant advised transferring the patient to a hospital in Baltimore.

On admission to University Hospital at 4:30 P.M., February 9, the patient appeared quite toxic, moderately jaundiced, her temperature was 99.8° F., pulse 128, respiration 28 and blood pressure 185/108. An abdominal examination revealed the height of the fundus to be 31 cm. The back could be felt in a right occipital anterior position. However, the abdomen was moderately tense and palpation of the fetus, especially the head, was quite difficult. There was some question as to whether the fetal heart could be heard. She was given pantopon gr. ⅓ and 1,000 cc. each of 5 per cent glucose and normal saline. One hour later she was referred for a roentgen examination of the abdomen. This revealed a markedly distended abdomen, the presence of a single fetus in transverse position, the fetal head very large and lying in the right iliac fossa. Gas was present within the fetus conforming to the distribution of the liver vessels, heart and aorta. A large mass was present in the mid upper abdomen, the right border of it being well defined. An impression

FIG. 1. Case 1. Anteroposterior roentgenogram of the abdomen showing a large mass (uterus) in the upper abdomen. The right border lies just medial to the distended ascending colon. Hydrocephalic fetal head overlying right ilium and sacrum. Paravertebral mass (retroperitoneal hematoma) to the right of eleventh and twelfth thoracic vertebrae.

of a hydrocephalic fetus and fetal death was made. The exact nature of the mass was not determined but was thought to be associated with the uterus (Fig. 1 and 2).

At 8:00 P.M. the patient was given a general anesthetic, the hydrocephalic fetus was removed from below by a destructive operation. Palpation of the uterus following delivery revealed a large rent in the anterior uterine wall extending from the cervix to the fundus. A laparotomy was then performed. Upon opening the abdomen the placenta was found free in the abdominal cavity with approximately 2,000 cc. of blood. The presence of a ruptured uterus was confirmed and the tear in the uterus appeared to be an old one. A supravaginal hysterectomy was done. The patient received 5 pints of citrated blood and 1 pint of plasma during the operation. The postoperative course was rather stormy and she died the third day after the operation.

Autopsy permission was granted and the findings were as follows: Traumatic, vesicovaginal fistula, intrapartum; retroperitoneal hematoma; hydronephrosis, hydroureter, right; hydrohematoperitoneum, 2,500 cc.; jaundice, skin and mucous membrane; fibrinous peritonitis, generalized; lower midline incision, recent; passive congestion, lungs; pulmonary infarcts, left multiple. The fetus was disposed of immediately after delivery, hence postmortem studies of it were not possible. An attempt to determine the type of gas present within the circulatory system of the fetus would have been helpful.

CASE II. R. S., a colored female, aged twenty-three, was first seen in the obstetrical clinic on July 15, 1946, at which time a diagnosis of pregnancy was made, duration approximately six months. As two blood Wassermann reactions were found to be strongly positive the patient was referred for rapid luetic therapy.

A past obstetrical history revealed one full term living child delivered in 1942 without complications. The patient visited the clinic at rather regular intervals and on September 30, 1946, for the first time the fetal heart sounds were not heard. On this same date the abdomen was found to be rather large and out of proportion to the duration of pregnancy. Polyhydramnios was suspected and the patient was referred for roentgen examination. This revealed a fetus of approximately eight months' duration with suggestive overlapping of the cranial bones. The uterus seemed quite large, larger than normal for the fetal size, and a

FIG. 2. Case 1. Lateral roentgenogram of the abdomen demonstrating gas in the fetal circulatory system. The large collection is within the heart. Other globules are noted in the thoracic aorta and abdominal vessels.

roentgenological impression of hydramnios and probable fetal death was made. No gas was noted in the fetal circulatory system. On October 11, 1946, the patient was admitted to the hospital because of slight vaginal bleeding. As roentgen placental visualization is done routinely on patients bleeding in the last trimester, the patient was again referred for roentgen examination. At this time well demonstrated gas was noted in the vessels of the liver, great abdominal vessels as well as the heart. The fetus assumed a rather relaxed position, the uterus was very large and the placenta was located in the anterior fundus. The fetal long bones were well demonstrated and practically all showed areas of marked decalcification adjacent to the epiphyses, with slight condensation of bone on the opposing surfaces. An impression of definite fetal death was made. The changes in the fetal long bones were thought to be due to lues (Fig. 3). Within the next few hours the patient went into labor. Labor was rather prolonged, lasting thirty-nine hours and twenty-two minutes. A 7 pound dead fetus was delivered. The puerperium was entirely normal and the patient was discharged from the hospital nine days later.

Fig. 3. Case II. Lateral view of the abdomen demonstrating gas in the fetal circulatory system: heart, abdominal vessels and umbilical cord. The uterus is quite large and the fetus assumes a relaxed position due to hydramnios. The placenta is attached to the anterior uterine wall.

Fig. 4. Case II. Anteroposterior view of the fetus immediately after delivery showing gas in the circulatory system similar to Figure 3. (Film in reverse position.)

A roentgen examination of the fetus after delivery showed similar distribution of gas as previously described as well as a clearer demonstration of the bone changes (Fig. 4 and 5). A postpartum examination of the fetus showed marked maceration with generalized autolysis of the viscera. An attempt was made to collect gas from the vessels for study but due to the small amounts present and technical difficulties encountered none was obtained. A section of a femur was removed for study but the pathologist was unable to find any definite abnormality. The cause of death was attributed to lues.

Case III. M. W., a colored primipara, aged thirty-two, was admitted as a registered service patient on February 26, 1947, at term, not in labor, and with the membranes ruptured since 4 P.M. on February 25. The patient was admitted with a diagnosis of premature rupture of membranes for normal, routine delivery.

FIG. 5. Case II. Showing bone changes in lower extremities of the fetus. Zones of decalcification with adjacent increased bone density in the ends of the long bones.

Her family and past histories were non-contributory. The prenatal course was essentially normal with the exception of a large granulomatous lesion of the right and left labia which was diagnosed as granuloma inguinale in the skin dispensary where she was followed throughout her pregnancy. On admission to the hospital the urine was negative; hemoglobin 90 per cent, blood pressure 104/56, temperature 99.8° F., respiration 20, pulse 88. The fetus was in the left occipito-anterior position with the presenting part fixed. Fetal heart sounds were heard in the left lower quadrant. On February 27 the patient's temperature rose to 102.6° F. The white blood cell count was 14,200, the urine was negative. A diagnosis of antepartum infection was made at this time and the patient was placed on routine chemotherapy (penicillin and sulfadiazine). Sulfadiazine was discontinued on February 28 because of a developing anuria. The blood pressure still remained normal. The fetal heart sounds were last heard at 1 P.M. on February 28. Although the temperature improved in the evening of that day, the patient appeared toxic and routine parental fluids were started. At 1 A.M. on March 1, the patient was acutely distended. Gastric suction was started and a temporary diagnosis of paralytic ileus was made. The patient did not improve and the temperature gradually rose to 102° F.

A roentgen examination made on the afternoon of March 1, revealed a large amount of intrauterine gas causing marked distention of the uterus and separating the uterine wall from the fetus (Fig. 6 and 7). Similarly there was gas seen throughout the tissues of the fetus: intracranial, intrathoracic, intra-abdominal, subcutaneous and intramuscular. The fetus appeared to be full term, in right occipito-anterior position and obviously dead. Following this information supportive measures were continued.

An emergency blood chemistry on March 1 showed urea nitrogen of 23. A rectal examination revealed the cervix one finger tip dilated, the presenting part 1 cm. above the spines and the cervix 25 per cent effaced. The patient gradually grew worse and her urea nitrogen rose to a maximum of 77 on March 5. A pelvic examination on the night of March 3 revealed crepitation of the cervix. On March 4 a sterile pelvic examination was repeated, amniorrhexis performed and a scalp clamp applied. On March

FIG. 6. Case III. Anteroposterior view of the abdomen, showing large amount of intrauterine gas and gas throughout the tissues of the fetus.

5 the patient finally began to void and she began to improve slightly, her urea nitrogen being 62 and uric acid 12.8 the following day. Uterine cultures on March 3 revealed microaerophilic gamma streptococcus. The urine culture of March 1 was negative. On March 6 at obstetrical conference a decision was made to deliver the patient from below in view of the optimum improvement. Accordingly, on March 6 the patient was given a general anesthetic and delivered from below by a destructive operation. The fetus on delivery was considerably macerated and decomposed and the umbilical cord had completely sloughed away. (Fig. 8 is a roentgenogram of the fetus immediately after delivery.) During the procedure approximately 2,000 cc. of foul smelling pus and decomposed debris escaped from the uterine cavity along with a quantity of equally foul smelling gas. Exploration of the uterine cavity after delivery revealed no tears. Cultures taken at the time of delivery from the placenta and baby revealed pure cultures of *B. coli* in heavy growths.

Following delivery the temperature remained around 102° F. and returned to within normal limits on her second day. However, there were some remissions and exacerbations of her temperature, the high being 101° F. on her fifth and sixth postpartum days. This was associated with foul, scant lochia which when treated with chemotherapy, ergotrate and Dakin's solution to the perineum q.i.d., gradually returned to normal. The patient was allowed up on her seventh postpartum day and was discharged in an improved condition on March 16, 1947, her tenth day, with a diagnosis of (1) puerperal endometritis, (2) stillbirth, cause of death—intrauterine asphyxia, secondary to intrapartum infection.

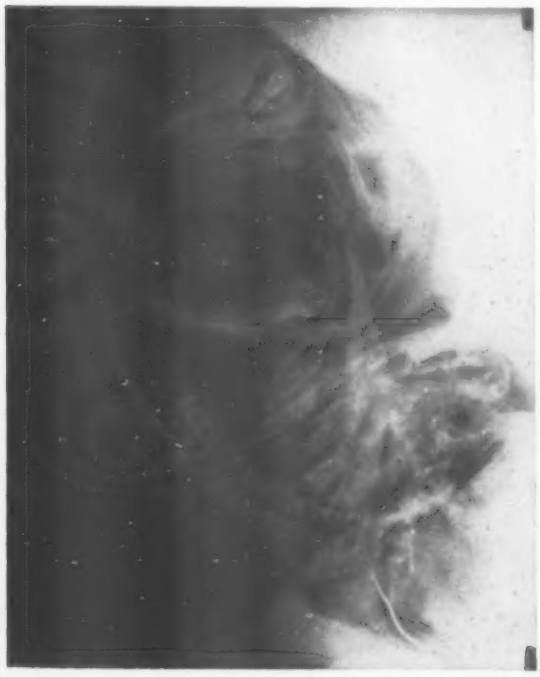

Fig. 7. Case III. Lateral view of the abdomen, showing distention of the uterus by gas and better visualization of gas in the tissues of the fetus.

Fig. 8. Case III. Fetus immediately after delivery, demonstrating gas throughout all tissues.

DISCUSSION

Cases I and II demonstrate very well the presence of gas in the fetal circulatory system. Attention was first called to the roentgen demonstration of this condition by Roberts[1] in 1944. He reported 2 cases both showing similar distribution of gas in the fetal circulatory system. He contributed this as a new sign of fetal death. He gave no explanation as to the source or cause of the gas but stated that further investigation along this line would follow. In the 2 cases presented here likewise no definite cause for the gas was found. Presumptive evidence would lead one to think

that it is due to decomposition of blood elements. The first fetus was destroyed before any studies could be made and attempts to do the same on the second fetus were unsuccessful. Certainly the presence of gas in the circulatory system must be a late sign of fetal death.

Although many articles have appeared in the literature concerning the diagnosis of uterine rupture, little has been mentioned regarding the roentgen observations. Hoffman,[2] in one of his case reports, makes this statement: "X-ray examination revealed the fetus high in the pelvis with a soft tissue tumor filling the pelvis, separate from the fetus. The diagnosis was 'ruptured uterus.' "

Failure to include a roentgen examination in this condition may be easily attributable to the fact that most patients with uterine rupture are readily diagnosed clinically by the obstetrician and the condition is treated as an emergency. Time consumed by roentgen studies would further endanger the life of the patient. However, there is a small group of these cases where the diagnosis is not very clear-cut, perhaps only suggestive, or not evident at all. Here, a roentgen examination may prove invaluable, especially if the tear in the uterus is great enough to permit the fetus to enter the abdominal cavity. The roentgen findings, as demonstrated in this case, are:

1. The presence of a well defined mass in the abdominal cavity, the contour of which suggests a contracted uterus.
2. The fetus outside the confines of this mass.
3. The absence of a uterine wall circumscribing the fetus.
4. Unusual position of the fetus.

Another change which may have been present but not demonstrated is the presence of free gas in the peritoneal cavity. Although the diagnosis of uterine rupture was not suggested at the time of the initial roentgen interpretation, a review of the case brought out the absence of a uterine wall circumscribing the fetus.

The third case is one of uterine distention with gas associated with generalized fetal gas due to infection. The organism producing the gas is an anaerobic streptococcus. Jordan and Burrows[3] state that this type of organism has been found producing puerperal sepsis and is gas forming and usually non-hemolytic.

SUMMARY

Three cases are presented as follows:

1. Uterine rupture with roentgen manifestations of the same, associated with evidence of gas in the fetal circulatory system.
2. Fetal death in utero demonstrated by gas in the fetal circulatory system.
3. Puerperal sepsis with roentgen manifestations of gas in the uterus and fetus, the gas being produced by a gas-forming organism.

CONCLUSIONS

1. The inclusion of roentgen examinations in cases of questionable ruptured uterus may prove helpful in arriving at a diagnosis.
2. The fetus in utero should be inspected closely for the presence of gas in the circulatory system. If present it indicates fetal death.
3. Roentgen examinations in cases of abnormal abdominal distention associated with puerperal sepsis may prove useful in eliminating other causes for distention.

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SARCOMA OF THE URINARY BLADDER IN CHILDREN*

WITH A REVIEW OF THE LITERATURE

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TUMORS of the urinary bladder in children are rare and nearly always malignant in nature. Not only are few found in the literature, but almost all are reports of single cases. Six authors have reported 2 cases each and none have reported more than this number. Most pediatricians have not seen a bladder tumor, and few urologists or radiologists have had experience with this type of neoplasm in children. For this reason a report of 2 additional cases with a review of the literature would seem to be timely.

Eighty-nine cases of malignant bladder tumors in children under ten years of age were found in the literature. Deming[6] reviewed 55 cases in 1924, and of Crane and Tremblay's[5] cases in 1943, 20 were in this age group. Fourteen others have been reported since. Adding the 2 cases reported in this paper to those from the literature, the total is brought to 91 cases.

PATHOLOGICAL FINDINGS

Malignant bladder tumors in children are all of mesodermal derivation. A few benign papillomas have been reported, but no authenticated instance of carcinoma. In Table 1 are listed the various types of sarcomas of the bladder found in the literature. According to Ewing[9] they may be classified histopathologically as angio-, chondro-, fibro-, glio-, lipo-, myo-, myxo-, neuro-, and osteo-sarcoma. Approximately 60 per per cent of the cases were found to be myxosarcoma, myxoma, or classified as just sarcoma. Many of the latter would probably fall into a more specific group if they were reviewed in the light of the classification of the present day. The histopathological picture in each form varies as to the type of cell present. It is beyond the scope of this paper to discuss the microscopic details of each, but they all present a picture of an embryonal sarcoma with variations according to the dominant cell types (Deming[6] and Ewing[9]). The myxosarcoma, for example, is a rather cellular structure with round or spindle-shaped cells which are suspended in a homogeneously staining material. This background material is not unlike the gelatinous substance found in the umbilical cord. In it, blood vessels are thin walled and are often numerous.

TABLE I

TYPES OF SARCOMA OF THE BLADDER AS FOUND IN THE LITERATURE

Type	Cases	
	No.	Per Cent
Sarcoma	28	31.0
Myxoma	15	16.4
Myxosarcoma	11	12.1
Rhabdomyosarcoma	9	9.9
Fibrosarcoma	8	8.7
Lymphosarcoma	4	4.3
Rhabdomyomyxosarcoma	3	3.3
Leiomyosarcoma	3	3.3
Fibromyxosarcoma	2	2.2
Rhabdomyoma	2	2.2
Spindle cell sarcoma	2	2.2
Embryoma	1	1.1
Neurogenic sarcoma	1	1.1
Osteogenic sarcoma	1	1.1
Plexiform neurofibrosarcoma	1	1.1
Total	91	100

These tumors are always highly malignant despite the fact that they rarely metastasize. If metastasis does occur, it is

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late. Wheelock,[30] in a discussion of sarcomas of the bladder which included and stressed adult cases, states that 23 per cent metastasized to the aortic nodes, liver, and lungs. Even this relatively low figure is not borne out in children, possibly because of their early death. Deming emphasizes the infrequent metastases in his series of 55 cases. Crane and Tremblay[5] list only 3 cases in children that had metastases, 1 to the periotoneum and 2 to the lungs. No mention has been made of metastases in any of the other reports of cases in the literature although autopsy examinations were recorded in most instances. In contrast, these sarcomas invade by direct extension. The neighboring vagina, uterus, prostate, pelvis or abdominal walls are involved with a startling rapidity. The ureters are often occluded at the vesical orifice, but extension of the tumor to the upper ureters has not been described. These lesions all grow rapidly and consistently recur following removal.

Grossly the vesical sarcomas are usually soft, friable, vascular, and hemorrhagic. They vary from polypoid, pedunculated, sessile, to massive in size and shape. Their surface is smooth and glistening, and except for occasional ulcerations these neoplasms are covered with bladder epithelium. Rhabdomyomyxosarcomas have consistently been described as a "bunch of grapes" or like a hydatidiform mole. Otherwise the gross appearances are not characteristic. These lesions have usually infiltrated beyond their apparent visible or palpable limits. In Case 11 the whole bladder wall was microscopically involved, despite its grossly normal appearance. Similar findings were present in several of the cases reported in the literature.

The location of the neoplasms in the bladder is highly pertinent. They involved the trigonal area or were widespread throughout the bladder in 65 per cent and involved the walls only in 35 per cent of the cases. Many of the latter were also located near the urethral or ureteral openings. This basal distribution makes complete surgical extirpation difficult or impossible short of a cystectomy.

The age and sex distribution are given in Table 11. Seventy-five per cent occurred in children five years old or younger, and 40 per cent in infants two years or under, indicative of a definite predominance in younger children and infants. The youngest case occurred in a three day old boy.[14] Of the 91 cases, 58 were in males, 19 in females, and in 14 the sex was not stated.

TABLE II

AGE AND SEX

Age, Yr.	Male	Female	Sex Not Stated	Total
Under 1	7	1	4	12
1	6	4	1	11
2	11	1	3	15
3	6	2	3	11
4	8	4	1	13
5	5	3	0	8
6	7	1	0	8
7	3	2	0	5
8	0	0	0	0
9	3	0	0	3
10	2	0	1	3
A child	0	1	1	2
Total	58	19	14	91

These vesical sarcomas showed a preference for boys in a ratio of three to one.

The predilection for the infant would suggest the congenital or "cell rest" theory of pathogenesis. There were no other findings which would further substantiate the varied hypotheses formulated to explain the development of these tumors. One factor of importance, however, has been the involvement of the bladder only, in many cases without extravesical tumor, which rather disputes the theory that the sarcomas arise solely from the perivesical tissues, as suggested by some authors.[22]

SYMPTOMATOLOGY

The early symptoms were predominantly those due to vesical irritation: frequency, dysuria, urgency, incontinence, or enuresis. Hematuria occurred in a large majority of the reported cases, at times

early but usually late. Most often bleeding was intermittent but occasionally it was gross and excessive. Pain was also variable in degree and type, but usually it occurred in a suprapubic site and was worse on micturition. Infection, with pyuria, was prone to develop early. This often resulted in a diagnosis of uncomplicated cystitis, with a resultant delay in further diagnostic studies. The great majority of cases were not seriously considered until urethral obstruction intervened. The time such obstruction appeared was variable, and frequently it was the presenting complaint. A large suprapubic mass appeared with a regular frequency, either from a distended bladder, actual tumor mass, or both. Following these vesical symptoms, which sometimes were present for a few days to months, renal infection and failure appeared and symptoms of these dominated the picture thereafter. In other instances, the latter were the initial symptoms. The course was then rapidly retrogressive, with uremia, systemic infection, and death.

DIAGNOSIS

The history indicated involvement of the urinary tract. Abdominal palpation often revealed a suprapubic mass, the bladder distended with urine and (or) tumor. Bimanual rectal palpation was informative, a procedure too often neglected because of the child's or the parents' objections. Urinalysis showed the presence of infection with bacteria, white blood cells, and usually red blood cells. Fever and an elevated white blood cell count were almost constantly present. Roentgen examination ruled out opaque calculi and in one case showed a mottled calcification due to calcareous deposits on a slowly-growing myxoma.[18]

Urographic and cystographic studies could, as a rule, establish the presence of a tumor. Cystography using air instillation, followed by contrast medium, was more satisfactory. The neoplasm when contrast medium was employed caused a filling defect, usually basal in location, and often multiple. With air and profile views the tumor was frequently directly visualized. Local or generalized irregularity of the walls plus a small bladder capacity implied infiltration of the whole bladder. Ureteral reflux occasionally was found with ureterectasis. Cystography proved to be a safe and simple procedure at all ages. It involved essentially no risk, and it is desirable that cystography precede cystoscopy. Caliectasis, pyelectasis, and ureterectasis, with poor kidney function, were often demonstrated with urographic studies, as well as deformity of the bladder, displacement, and filling defects. Non-visualization was not uncommon due to poor kidney function or (and) excess gas shadows, especially in the infant.

Cystoscopic examination was confirmatory and some tumors were found that were not otherwise visualized. In several instances, however, this procedure caused severe hemorrhage.[10] Occasionally it was impossible to outline the tumor or differentiate it from prostatic enlargement. [1,28] In Case 1 cystoscopy led to a definite delay in treatment as negative results were obtained on three separate occasions. A specimen for microscopic study could usually be obtained during cystoscopic examination. This procedure proved to be essential in excluding benign tumors, such as angioma, fibroma, myoma, dermoid, and papilloma, which presented the same gross appearances as the sarcomas. The benign tumors were more uncommon.[4] Deming found an incidence of 20 per cent. Cystotomy was at times necessary to secure material for histopathological study.

PROGNOSIS

The prognosis was uniformly grave. The very rapid rate of growth often resulted in early urethral or ureteral obstruction with kidney damage and infection before the diagnosis was suspected. The pyelonephritis and pyelectasis were followed by uremia, cachexia or intervening systemic infection, and death. The average case died within six months, and survival of more than a year was unusual. These early and

rapid deaths occurred despite surgical intervention and extirpation of the neoplasm. Recurrence after excision was not only the rule, but surgical trauma seemed to act as a stimulus and increased the rate of growth.

Uhlmann, Grossman and Calvin[26] reported a case of a sarcoma which was excised from the bladder of a ten year old boy. In one week it had recurred to the extent that it was protruding from the operative site.

viving two years.[12] Feggetter's case had lived one year when reported. A personal communication reveals that she is now a healthy young woman, twenty-two years of age, with an apparently normal bladder, twelve years after treatment. Of interest is the fact that she married and conceived, despite her roentgen therapy. Evidently the ovaries were not irreparably damaged.

TABLE III

CASES LIVING AT THE TIME OF THEIR REPORT IN THE LITERATURE

Author	Date	Type	Therapy	Course
1. Deschamps[7]	1791	Fibrosarcoma	Resected	Alive
2. Klein[16]	1895	Polypoid sarcoma	Resected	Alive
3. Mixter[20]	1917	Embryoma	Tumor and base excised	Well at 2 years
4. Feggetter[10]	1937	Sarcoma	Tumor and base excised plus deep roentgen therapy	Cured, 12 year survival
5. Hess[11]	1938	Plexiform neurofibrosarcoma	Partial resection and some roentgen therapy	Alive 7 years with disease
6. Meade[18]	1943	Polypoid myxoma	Tumor and base excised	Alive and well
7. Tahara, Burt and Hess[25]	1945	Fibromyxosarcoma	Resection plus deep roentgen therapy	Well at 3 months*

* Died after surviving 2 years.[12]

The following cases were alive at the time they were reported. Hess's plexiform neurofibrosarcoma was living at seven years with known residual disease. It undoubtedly was a slowly growing lesion and it did not behave in a manner comparable to the other cases. The short follow-up period in 5 of the other cases and also in Case 11 in this paper was not entirely satisfactory. Cases 1, 2, 3, and 6 (Table III) were located high on the bladder wall or in the fundus, which permitted wide excision through healthy tissue. As a result they may have resulted in actual cures. The study of the other reported cases with similar excision, however, does not confirm this favorable prognosis. The 2 remaining cases, Case 5 (Feggetter)[10] and Case 7 (Tahara, Burt and Hess)[25] in Table III, in addition to excision, received deep roentgen therapy. Tahara, Burt and Hess's case, though well at the time of its reporting, died after surIrradiation seems to have delayed recurrences in Tahara, Burt and Hess's case, though the patient ultimately succumbed. In the case reported by Feggetter the combination of excision and roentgen treatment resulted in the only conclusive cure reported in the literature.

If 6 of these cases are considered cures, the mortality rate was 93 per cent. The actual rate was probably higher than this when the short follow-up period and the unreported cases are taken into consideration. From this review, except for the one case above, no mortality rate can be given in terms of five year cures with a tumor-free patient. Sarcoma of the bladder is one of the most malignant tumors that occur in children.

THERAPY

The early cases in the literature had excision or curettement of the tumors with and without excision of the bases. The great majority in the last half century were treated by excision and, where possible,

resection of adjacent, presumably normal, bladder tissue. In lieu of the latter, cautery of the base was performed in lesions located in the trigone. Radium implantation was occasionally added. Suprapubic drainage was the rule for urethral obstruction. With these methods, fulminating recurrences developed with a regular and distressing frequency. An example is the case of Welfeld, Hill and Hillebrand.[28] A rhabdomyomyxosarcoma was "pulled" from the bladder wall near the urethral orifice. Three days later when the suprapubic catheter was removed, the tumor had again filled the bladder. The neoplasm then bulged out through the excision, and the external mass reached the size of a grapefruit in ten days.

Because of these poor results, Campbell,[3] Deming,[6] Tahara, Burt and Hess,[25] and many others, have condemned excision, except for the occasional isolated tumor located near the fundus where a wide border of healthy tissue can be included. Multiple biopsies of the remaining "normal" bladder to exclude microscopic infiltration of the other walls and base can be performed. Postoperative radiation therapy should follow.[3,5,25] In the typical cases with a trigonal location of the sarcoma, radical methods are recommended. Deming[6] in 1924 suggested cystectomy as a possibility, but knew of no case in a child where it had been tried. Crane and Tremblay[5] stated that cystectomy, radium, and roentgen therapy offered the only promise of alleviation or cure. Cecil,[4] Campbell,[3] Ratliff and Valk,[23] Weyerbacher and Balch,[29] and others, stated that complete cystectomy was the therapy of choice. Khoury and Speer[14] performed a cystectomy on a five and a half month old child who had symptoms from the age of three days. The ureters were transplanted to the skin, the bladder removed, and later the ureters placed in the sigmoid. The patient died six and a half months later with kidney failure and infection. Despite ultimate death, the operation was a success in that the patient withstood the procedure statisfactorily. Kidney damage and infection in this case was too great before the operation was undertaken. Ritter and Kramer[24] stated that the operative shock should be no more that that from a prostatectomy in an adult, and it should not be considered an impossible or too formidable procedure. The general opinion of the writers in the literature concurred in this method, and stressed the need for the procedure as soon as the diagnosis was established. Their decisions for cystectomy were unfortunately formulated after the autopsy, and as a result it has had relatively little trial in children.

A comparatively small number of cases have had roentgen therapy. Radon seeds have been used in several cases to the base of the excised tumors.[3,6,19,23] This type of irradiation has proved unsatisfactory, largely because of the extent of the neoplasms. Teleradium and deep roentgen therapy have been used in a small number of patients. Rhabdomyomyxosarcoma and spindle cell sarcoma are radioresistant but do show some response in that they may be temporarily held in check.[14,21,26] Sarcoma, myxosarcoma, fibromyxosarcoma, fibromyxoma, and myxoma have all been shown to be more responsive and appear to be moderately radiosensitive.[1,3,5,6,10,11,14,25,27,29] Tahara, Burt and Hess, who had a living patient at the time of their report, gave 2,100 roentgens anteriorly and 2,400 r posteriorly, measured in air, as a postoperative course. Hess believes radium and roentgen irradiation should be of great therapeutic aid and strongly recommends its use. Crane and Tremblay, Khoury and Speer, and the above-mentioned writers, feel that these rapidly growing neoplasms are definitely sensitive enough to give promise with roentgen therapy. Kikuti and Minakawa[15] reported an ovarian fibromyxosarcoma completely destroyed locally by roentgen treatment, though metastases appeared elsewhere eight months later. The dosage used was not given. In Case 11, as reported in this paper, it is difficult to assay the effect of the roentgen treatment as it was always given in combination with excision. Nevertheless, the sarcoma seemed to have

been held in check somewhat for two months on two occasions, and from the cases in the literature with excision alone, one would not expect relief for this short period.

In a discussion of the most beneficial therapy the behavior and characteristics of this group of neoplasms must be recalled. Metastases occur rarely and late. This reluctance to metastasize offers an opportunity for treatment not found in the usual malignant condition. The exceedingly rapid growth, local invasion, recurrence following resection, early interference with kidney function plus infection dominate the picture. A multiple attack is required to meet this, and in a favorable case it can be attempted.

In the uncommon, isolated, and localized fundal sarcomas, wide excision, multiple biopsy of the remaining bladder wall, and deep postoperative roentgen therapy are practical and desirable. The patients must be followed closely with repeated cystograms and occasional cystoscopic examinations. In the typical case with infection, obstruction, and a trigonal sarcoma, excision is contraindicated. Antibiotics, suprapubic cystotomy for drainage, and general systemic care should be instituted and are necessary to sustain life. As soon as the diagnosis is established, and the child is no longer acutely ill, deep roentgen therapy can be instituted. Deep roentgen therapy should be given to skin tolerance through at least two ample ports. Damage to testicular, ovarian, or bony epiphyseal tissues can be disregarded. A depth dose of 3,000 r would seem desirable. Early radiation treatment is preferable before contemplating surgical procedures. Since genitourinary tract infection and renal damage are usually present, radical surgery, if not entirely contraindicated at this time, will certainly add to the strain on the child and is best delayed. In addition, ureteral transplantation in the presence of infection is doomed to failure, but may be performed later with more hope of success.

Following treatment with general measures and radiation therapy, careful evaluation of the patient's condition can be made. The amount of kidney damage must be accurately determined. If definite renal failure is present and has persisted, palliative measures only need be considered. If kidney function is satisfactory and infection controlled, further treatment of the sarcoma may be feasible. The neoplasm will possibly have been reduced in size, in accordance with cases reported in the literature. The relatively resistant types of sarcomas will most likely have been held in check and the others will have shown varying degrees of regression and partial destruction. Except for the lymphosarcoma, which will grossly have disappeared, this amount of radiation will not cause a complete disappearance of the tumor. Roentgen treatment alone cannot result in the entire destruction of these neoplasms. For this reason a complete cystectomy should follow. This is a radical form of therapy for an adult, and especially so in an infant or a child. However, since invariable failure resulted from other operative procedures, cystectomy offers the only hope for a cure. Cystectomy is being performed successfully in adults, and Khoury and Speer[14] have demonstrated its feasibility in an infant under adverse conditions. They recommend ureteral transplantation to the skin, cystectomy including the prostate and proximal urethra, and at a later date, transfer of the ureters to the sigmoid. Obviously it is no small matter to doom a child to a lifetime without a urinary bladder or to subject him to such a major operative procedure, but it offers him his only chance of relief from an otherwise hopeless lesion. Two to four months following operation, a second series of roentgen treatments can be given.

With the mortality rate of over 90 per cent for sarcomas of the bladder as a whole and 100 per cent for trigonal lesions, one cannot become optimistic over any method or plan of therapy. Despite this, and the many certain failures that can be expected from such a formidable neoplasm, the lit-

erature has conclusively demonstrated that therapeutic measures short of those discussed here have invariably met with failure.

REPORT OF CASES

Case 1. J. S., a colored male, aged twenty-one months, was admitted on May 4, 1946, to the Children's Hospital with a past history of six weeks' duration of progressive frequency, dysuria, straining, and a mucoid discharge from the urethra. He passed bright red blood the day before admission.

Physical examination, including abdominal and bimanual rectal palpation, was negative except for an inflamed glans penis. The urine showed white blood cells, red blood cells, and a positive culture for *Bacillus coli*.

A urogram was unsatisfactory. A cystogram demonstrated a space-occupying lesion, typical of a bladder tumor (Fig. 1, *A* and *B*). Cystoscopic examination was negative, though not entirely satisfactory due to blurred lenses. The patient was then signed out of the hospital against advice.

On readmission six weeks later, there was a marked increase of the previous signs and symptoms, plus fever and chills. The child was obviously seriously ill. The hematuria had continued intermittently. Visualization of a bifid left kidney pelvis and proximal ureter was obtained on urographic study and the bladder tumor was again demonstrated (Fig. 2). The latter had increased in size with apparent involvement of the entire organ. A suprapubic mass was easily palpable clinically. Two further cystoscopic examinations were also negative, though the operator was not entirely satisfied.

Suprapubic cystotomy demonstrated a lobulated, glistening, and hemorrhagic large tumor mass located largely in the trigone area, though it obviously involved the whole bladder. Biopsy only was attempted.

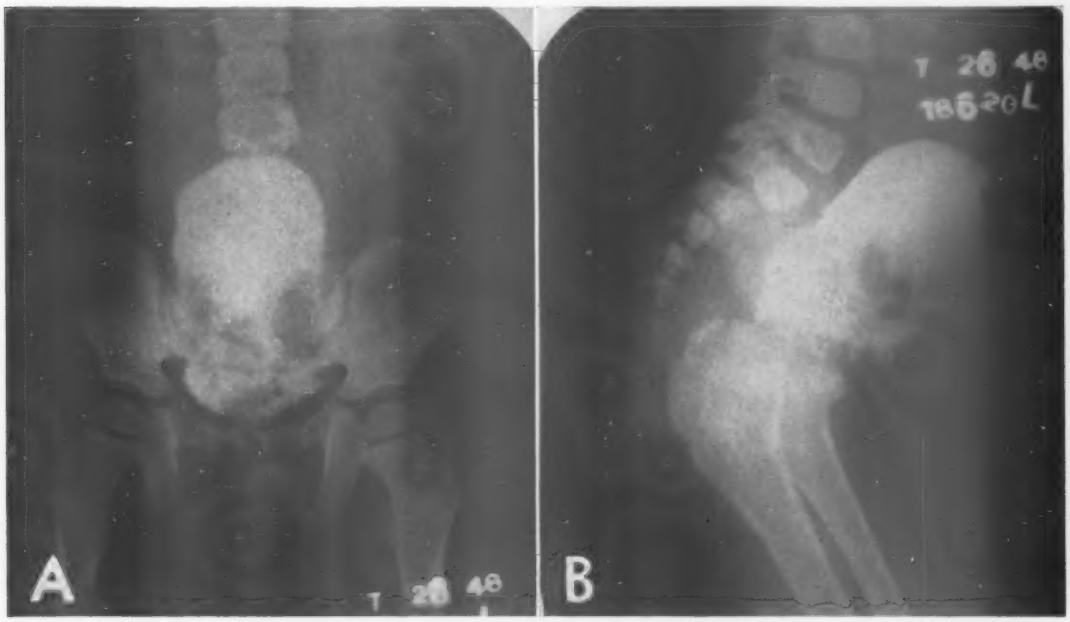

Fig. 1. Case 1. (*A*) Cystogram demonstrating a large filling defect typical of a neoplasm. (*B*) Lateral view.

The tumor cells on microscopic examination were round and oval, deeply staining, and in a myxomatous background, A few mitotic figures were present. Pathological diagnosis: Myxosarcoma (Fig. 3).

The patient was again signed out of the hospital against advice, and he died five months after his first admission. Death was presumably due to kidney infection and failure, which was present during his last hospital stay. No metastatic lesions were demonstrated during the period of observation by physical examination, roentgen examination of the chest and long bones, or exploratory operation.

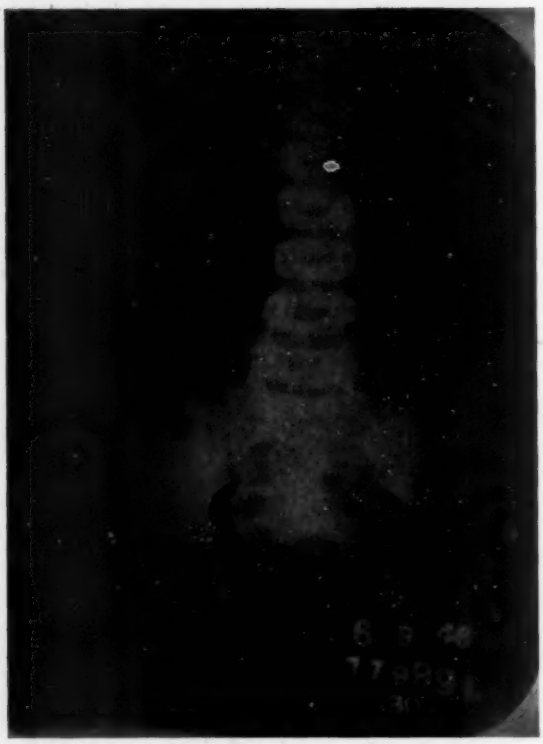

Fig. 2. Case I. Intravenous urogram showing a bifid left kidney pelvis and ureter as well as the myxosarcoma of the bladder.

Case II. C. H. (No. 064288), a white male, aged fifteen months, was admitted on June 24, 1947, to the Bryn Mawr Hospital with apparent abdominal pain of four hours' duration and a "lump" in the right scrotum. The patient had been voiding frequently and in small quantities for the past few weeks. Urinalysis and blood counts were within normal limits. A right hydrocelectomy and herniorrhaphy were performed following a five day observation period. The patient was discharged apparently well eight days after operation.

Two days latter the patient was readmitted with incontinence, dribbling, and abdominal pain which had commenced shortly after discharge from the hospital. Careful questioning revealed a story of difficulty when voiding for many months. Micturition had never been forceful and when attempted it precipitated bouts of fretting and crying. The bladder was palpable up to the umbilicus. A catheter when passed met an obstruction near the urethrovesical junction.

A subcutaneous urogram demonstrated slight dilatation of the right kidney pelvis and ureter and definite caliectasis, pyelectasis, and ureterectasis on the left. No dye was visible in the greatly distended bladder. A one hour film following catheterization and removal of 150 cc. of urine showed a definite decrease in the dilatation of the kidney pelvis. The bladder showed a bizarre pattern, partially due to collapse following removal of the large quantity of urine, spasm from inflammation and infection, a filling defect in the trigone area, and tumor infiltration of the walls (Fig. 4, *A* and *B*). On cystoscopy, a smooth, glistening, polypoid-like tumor was seen arising from the base. A cystogram demonstrated a filling defect at the same site (Fig. 5). A few bacteria and white blood cells were found in the urine. The white blood cell count was 13,600, the red blood cell count 4,490,000, the blood urea nitrogen 9 mg. Penicillin therapy was commenced and continued throughout the hospital stay.

A suprapubic cystotomy was performed and a soft yellow polypoid tumor with a broad base was excised. The base was fulgurated. Biopsy of the remaining bladder wall was performed, though it appeared normal except for some moderate thickening.

Pathological report: The sections showed a solid tumor mass consisting of closely packed neoplastic cells with little cytoplasm, large rounded or oval nuclei, and distinct nucleoli. There were numerous mitotic figures. No differentiation of any sort was seen, and there was no distinct connective tissue framework. Several areas showed a myxomatous background. Tissue from the bladder wall and fundus showed the same neoplastic cells. Diagnosis: Myxosarcoma.

Fig. 3. Case I. Photomicrograph, 100×. Myxosarcoma.

Roentgen therapy: A series of fifteen treatments was given through a 10 by 10 cm. portal over the bladder area using 200 kv., peak, 0.5 mm. copper and 1.0 mm. aluminum filter, 50 cm. distance, and 20 ma. A total of 880 r, measured in air, was given anteriorly and a similar amount posteriorly. The patient was discharged clinically well August 8, 1947.

Incontinence suddenly reappeared September 18, 1947, following a month and a half Christmas for a cystectomy. Roentgen irradiation, 720 r, was given through an anterior portal in fractional doses. Therapy was stopped the day before the following operation.

On December 28, 1947, the patient returned to the hospital. Urinalysis showed red and white blood cells and bacteria. The temperature ranged from 100 to 102°F. daily. Antibiotics were again given, and on January 5, 1948, exploration demonstrated no visible or palpable

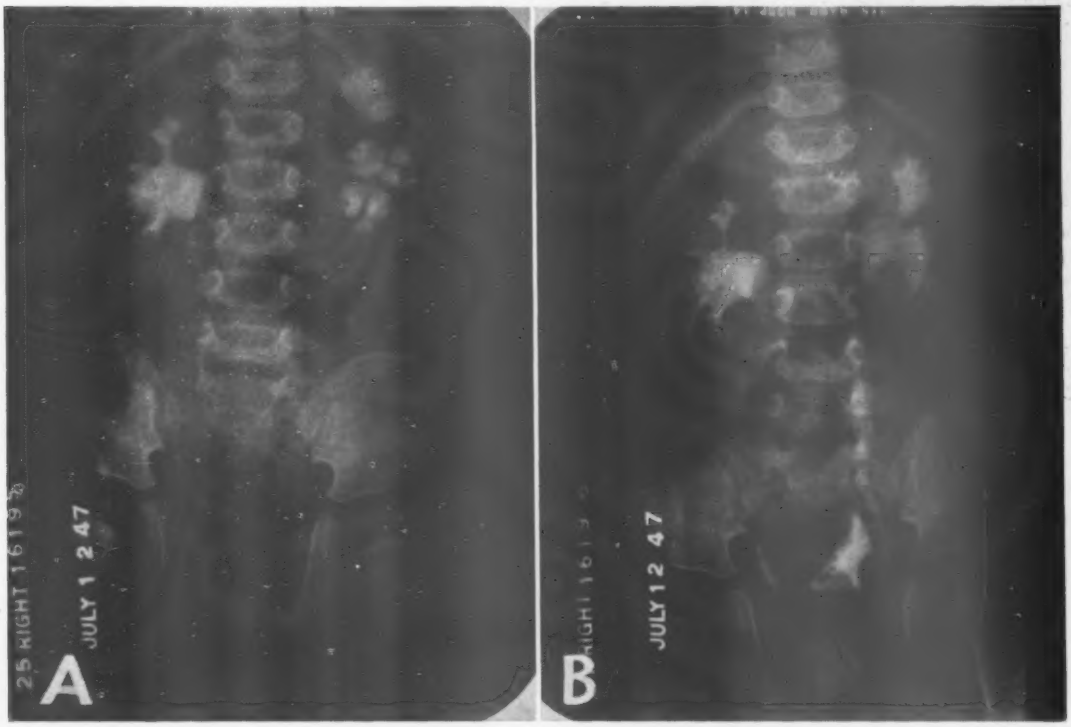

Fig. 4. Case 11. Intravenous urogram. (*A*) Caliectasis and pyelectasis, especially on the left. Bladder greatly distended. (*B*) A one hour film exposed after catheterization. The bladder presents a bizarre appearance due to collapse, neoplastic infiltration of the walls, and a filling defect at the base due to the tumor.

asymptomatic period. A recurrence was demonstrated by cystoscopic examination. At suprapubic cystotomy a nodular mass of myxomatous tissue was removed from the trigonal area. The base was fulgurated and thirteen radon seeds were implanted. The pathological report was similar to the previous description.

The patient was asymptomatic again for two months, after which time crampy abdominal pains recurred. Cystoscopy again showed a recurrence about the vesical orifice. The patient's symptoms were alleviated with sulfadiazine and streptomycin therapy, and he was discharged with instructions to return after tumor extension beyond the bladder. The right ureter was transplanted to the proximal portion of the rectum and the left to the distal portion of the sigmoid. Ureteral catheters were passed from the ureters out through a mushroom rectal tube. The postoperative course was stormy, and the patient's condition remained critical for a two week period. Intravenous administrations of plasma, blood, glucose and saline solution, plus streptomycin and sulfadiazine eventually controlled the infection, the chemical balance was re-established, and the anemia and hypoproteinemia were corrected. On February 6, 1948, a month following the

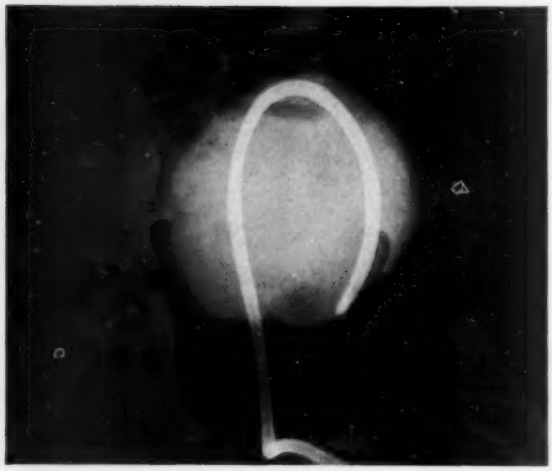

Fig. 5. Case II. Cystogram demonstrating the filling defect in the trigonal area.

ureteral transplantation, a total cystectomy was performed. This included the bladder, prostate, proximal urethra, and adjacent available tissues. The postoperative course was relatively quiet, compared to the previous operation, and recovery was rapid. A follow-up blood examination showed the urea nitrogen to be 15 mg., the uric acid 1.0 mg., and the blood protein, chlorides, carbon dioxide capacity, and blood counts were well within normal limits. The patient appeared to be well developed and clinically a healthy child two months following the cystectomy. A urogram at this time demonstrated caliectasis and pylectasis on the right and an essentially normal picture on the left.

Pathological examination of the bladder specimen showed no infiltration of the tumor cells into the prostate or to the other borders of the specimen. Apparently the lesion had been removed in toto. The tumor was firm, white, and uniform on cut sections. The original tumor was soft and myxomatous. The bladder wall in the specimen was obviously thickened throughout. Microscopic sections again showed dense infiltrations of tumor, cellular elements, largely in the submucosa layer. The cells were similar to those previously described, plus groups of oval, large, fascicular nuclei contained within a single elongated mass of cytoplasm. These latter formations of cells were undoubtedly myoid in origin. Diagnosis: Rhabdomyomyxosarcoma (Fig. 6, *A* and *B*).

Two and one half months following cystectomy a mass appeared in the left inguinal area and on bimanual palpation a lemon-sized suprapubic mass could be felt. These undoubtedly represented local recurrences in the regional lymph nodes. A roentgen survey of the long bones and chest was negative. Roentgen therapy is being given to these lesions, and they have all but disappeared with the course half completed. A depth dose of 3,500 to 4,000 r is to be given if tolerated by the patient. The prognosis is hopeless, but since there are no known distant metastases, a full course of roentgen therapy is planned.

Comment. Case I was obviously treated in an unsatisfactory manner. The family would not accept the diagnosis of a malignant tumor in so young an infant and they would not permit therapy of any type.

Case II, I believe, had excellent treatment except for two shortcomings. First

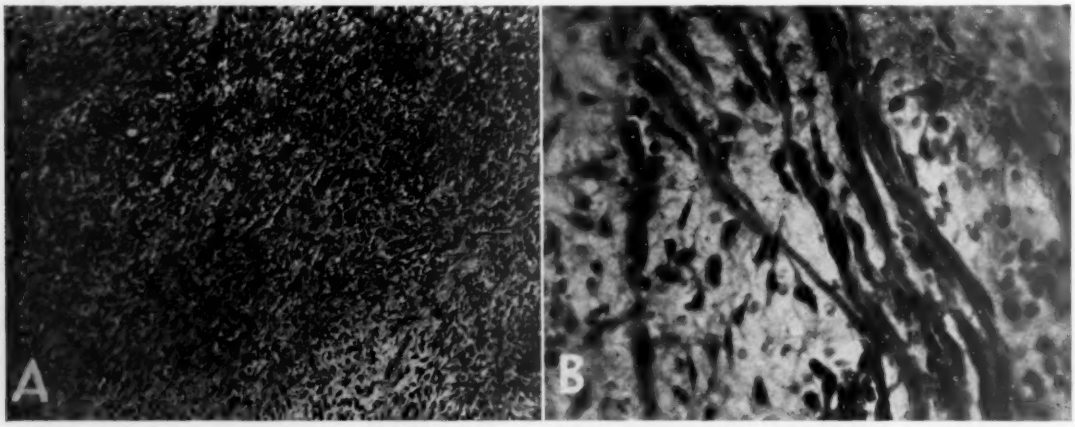

Fig. 6. Case II. (*A*) Photomicrograph, 100×. Rhabdomyomyxosarcoma. (*B*) 440×, showing the striated muscle cellular elements.

the amount of roentgen therapy was not large enough. Second, and more important, the ureteral transplantation and cystectomy should have followed the first course of radiation therapy. The diagnosis was established, the patient's condition was excellent, infection was controlled, and the kidney function was normal. Waiting was of no avail, except to prove, as do the cases in the literature, that these sarcomas recur following either excision, fulguration, radon seed implantation, and roentgen treatment, or a combination of these. In retrospect, all concerned with the patient feel this to be true. A cure might have been obtained had the cystectomy been done early. Though roentgen therapy is being given, complete destruction of the rhabdomyomyxosarcoma is hardly expected.

SUMMARY

Eighty-nine cases of sarcoma of the bladder in children under the age of ten were found in the literature. Two more cases are added. Sixty-five per cent were located in the trigone. Seventy-five per cent occurred in children five years old or under, and they were three times as common in males as in females.

Metastases occurred late and rarely. Local invasion and spread were rapid. Early symptoms were for the most part similar to those of genitourinary tract infection. Hematuria may occur early or late as does urethral and ureteral obstruction. Cystographic and urographic roentgen studies and cystoscopy demonstrated the presence of the neoplasms. Biopsy was necessary for exclusion of the occasionally found benign tumor.

The prognosis was grave; death usually occurred within six months. The mortality rate was 93 per cent with only one report of a five year survival without residual tumor.

Surgical extirpation appeared to stimulate the sarcomas and resulted in invariable and rapid recurrences, except for the less common lesions located and limited to the fundus.

Antibiotics, bladder drainage, and deep roentgen therapy are indicated as the first treatment of choice for the neoplasms located in or near the trigone. The great majority of these sarcomas are moderately radiosensitive and can be expected to show an appreciable response. Complete destruction of these tumors will not occur. If renal function is then found to be satisfactory, cystectomy should follow with ureteral transplantation and a second course of radiotherapy two to four months later.

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PARTLY DUPLICATED COLON*

CASE REPORT

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IN THE literature only a few cases of duplicated colons have been recorded. Weber and Dixon reported a case of an entire duplication, where the supernumerary colon reaching from the cecum to the rectum was situated parallel to the normal one. They both had a common mesocolon. There was no communication between the normal colon and the duplicated one; the latter was closed at both ends. Diagnosis was made by roentgen examination. The duplicatum appeared as a slight shadow. When examined with barium enema, many impressions caused by the duplicatum were visible on the normal colon. There was also found a duplication of the uterus. Another case was reported by Duhem and Monmignault, in which a girl, three years of age, had two transverse and descending colons, two rectums and two (functioning) anal canals. In this case also there was a duplication of the genital organs. Duplication of the colon was also reported by Asai, Ballance, Grohé, Ladd and Gross and Papayoannou. Gray reported a case of triplicated colon.

The following case is one of partial duplicated colon, running from the region of the hepatic flexure to the lower part of the descending colon. Proximally it ends blindly; the distal end communicates with the descending colon. A summary of the case history follows.

CASE REPORT

The patient was a girl, aged ten. Dull pains occurred in the right side of the abdomen. Spontaneous bowel movements only every third day. On the right side of the abdomen was a palpable mass, resembling a kidney in size and shape. This mass followed the respiratory movements and was easily moved with the hand. In another institution a roentgen examination was made, first with a barium enema and later with a "plain kidney film." This showed in the upper part of the right half of the abdomen a shadow identical with the palpated mass in size and shape. The shadow was calcium-like in density and granular in composition. In the opinion of this institution this shadow was probably caused by an extensive calcification of the right kidney.

The girl was then directed to our institution. We made a roentgenoscopic examination and afterwards a roentgenogram of the right kidney region (the latter in horizontal position (Fig. 1)). A shadow was found conforming to that of the earlier examination. In addition, we found that proximally the shadow was surrounded by a gascoat. We thought therefore

FIG. 1. Kidney-like shadow in the right part of the abdomen. The film was made after a barium enema examination. Gascoat proximally in the sac-like structure.

* The author observed this case in the Roentgen-Ray Department (Medical Chief: Dr. R. Holitsch) of the "Apponyi Poliklinik" in Budapest and subsequently published it in part in *Gyógyászat* (a Hungarian medical journal).

Fig. 2. Barium enema examination. There is no connection between the sac and the right part of the large intestine. A supernumerary colon runs to the descending colon.

that a sac-like structure was present connected with the gastrointestinal tract and that the shadow was caused by contrast material in the sac as a result of the barium enema examination. (The intestine-like structure running from the region of the under part of the shadow to the left part of the abdomen we thought then to be the normal transverse colon.) We had the patient take laxatives and purgatives many times in order to eliminate the contrast material. The picture, however, was unchanged, even after ten days. Then we performed a barium enema examination (Fig. 2). We could then establish the fact that the sac was not in connection with the proximal part of the colon. It was also obvious that the intestine-like structure, starting at the under part of the sac, was not identical with the transverse colon. This was shown to be in connection with the sac and identical with a supernumerary intestine, which was filled from the lower part of the descending colon and ran towards the right. It seemed probable that a supernumerary large intestine existed connecting the sac with the descending colon. Also there was found a large sigmoid.

Later we made an examination of the gastrointestinal tract with peroral filling. In the stomach, duodenum and small intestine, no pathological condition was found. Examination of the large intestine (24 hours p. c.) strengthened our earlier opinion (Fig. 3). Our findings were as follows.

There was a sac-like structure in the right side of the abdomen, which was connected with the descending colon by a supernumerary intestine. The sac-like structure was filling and evacuating only through this supernumerary intestine. We concluded that the interpretation

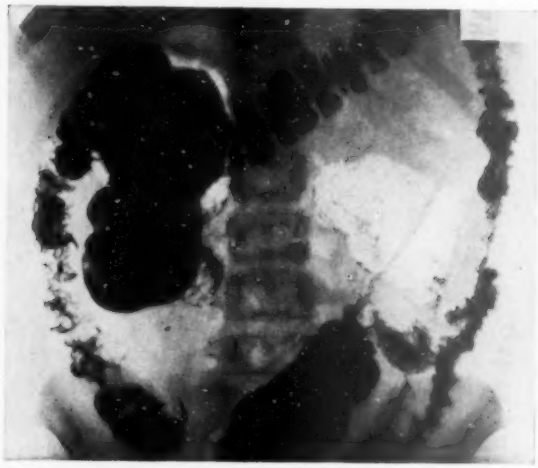

Fig. 3. Peroral filling after twenty-four hours p. c. The same findings.

in the first examination at the other institution was a diagnostic error.

After six months the patient was operated on. Roentgen examination made just before the operation showed the same findings as the earlier ones. The operation confirmed our impression. There was found a sac-like structure connected with the colon. A resection was not attempted because of the great risk involved. After ten years another operation was done (by Prof. Bakay, Klinik of the Pázmány Péter-University, Budapest). The important operative findings follow:* "... One can isolate only with difficulty the sac having the size of a child's head, because of the cicatrization caused by the earlier operation. The

* For placing this record at my disposal, I am indebted to Dr. A. Gusich, Adjunct of the Klinik.

structure is pliable and keeps the finger impressions. It is lying retroperitoneally and is situated between the upper part of the kidney, the flexura coli dextra and the liver. The sac continues toward the left behind the mesentery in a band the width of two fingers, having the appearance of an intestine. This intestine opens into the upper part of the sigmoid. In the course of the dissection of the sac—grown together with the vena cava and other organs—great veins were broken causing great bleeding. Also the sac wall was broken; then appeared a fecal-like mass. The bleeding veins and the ruptured sac were tamponated and the abdomen was left open. Peritonitis occurred, followed by death of the patient.

"From several parts of the sac excisions were made. Their microscopic examination showed a finding identical with that of the colon wall. The muscular layer was hypertrophied in some places." A developmental defect of the urogenital tract was not found.

On the basis of the microscopic examination the sac was that of an actual enlarged part of colon. I considered, therefore, the intestine starting from its under part and the sac as uniform structures and the sac itself as enlargement of this abnormal intestine. It exists a supernumerary colon as a developmental defect, which starts blindly at the region of the right flexure and opens into the descending colon. It is very probable that the enlargement developed secondarily. Golden reports that narrowings in the gastrointestinal tract occur as developmental defects. This kind of narrowing might exist in the opening of the sac, which caused difficult evacuation and therefore secondary enlargement. This narrowing was not proved anatomically (examination at the operation and section were not made from this point of view), but the great difficulty of evacuation indicates its presence. (The contrast material was not evacuated from the sac even after many months.)

Our case is substantially different from most of the cases reported because the supernumerary colon had its own mesocolon. Only Papayoannou reported an almost identical case. This was a girl, who had an immense tube-like structure connected with the colon. The whole structure was situated in an umbilical hernia. A bicarnate uterus was also found. I interpret Papayoannou's case in the same way as I do my own. In both cases there existed a supernumerary colon as a developmental defect, which secondarily partly enlarged. The fact that our patient had not also a development defect in the urogenital apparatus does not contradict this interpretation. The supernumerary colon opens into the descending colon so high that this part could not have a relation with the urogenital apparatus in the embryonic stage of the development.

SUMMARY

A girl, aged ten, had a sac-like structure the size of a child's head in the right part of the abdomen. Proximally this structure was closed; distally it continued as a supernumerary colon, which communicated with the distal part of the descending colon. This sac is considered to be an enlargement of the supernumerary colon.

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THE ROLES OF SURGERY AND RADIOLOGY IN THE TREATMENT OF CANCER*

By L. HENRY GARLAND, M.D.
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"Physicians are some of them so pleasing and conformable to the humour of the patient, as they press not the true cure of the disease; and some are so regular in proceeding according to art for the disease, they respect not sufficiently the condition of the patient." Of Regiment of Health. Francis Bacon, 1597.

INTRODUCTION

THE paradox of cancer remains that although our knowledge concerning the disease is steadily increasing, so are the number of cases, owing apparently to the fact that we are an aging population. No radiologist needs to be reminded of the fact that it is now the second most common cause of death in this country and that it ranks high as a source of morbidity. Although the general mortality trend is upwards, the specific mortality rate for certain types of cancer (such as those of the lip, cervix uteri, etc.) has taken an encouraging downward curve in the last decade, reflecting the results obtainable with more efficient or earlier treatment.[7]

The fact that the exact cause of cancer is still unknown tends to produce unwarranted pessimism on the part of many. In this connection it must be recollected that mere discovery of causation would not necessarily result in any precipitous or extraordinary improvement. The causes of tuberculosis and typhoid were discovered over fifty years ago, yet the cure of established cases of these two diseases still remains difficult. Their prevention is simplified, but their cure is still intricate. Similarly, when and if all of the causative agencies of cancer are found, it is anticipated that prevention—the real goal of medicine—rather than cure of established cases, will be greatly enhanced. In the meantime our main hope lies in early diagnosis, and prompt, adequate treatment.

Our principal weapons for the cure and palliation of cancer remain surgery and irradiation; subsidiary but sometimes important agents are chemotherapy, hemotherapy and miscellaneous medical procedures. The optimum method of treatment is often difficult to determine from reports in the literature, partly because of lack of uniformity in clinical staging of cases, and partly because of different methods of evaluating results.

It is not always of prime importance which of two given methods is used in an individual case: the essential thing is that the treatment be *adequate*. Sometimes this is effected equally well by surgery or irradiation; in others it requires a combination of both techniques and in still others surgery alone or irradiation alone is superior. Experience has taught us much of how and when to use these techniques. Today, I propose to review this experience and, in passing, to stress the advancing front of radiology. In doing so I would like to pay tribute to the distinguished surgeons, gynecologists and dermatologists who have played so great a part in blazing these therapeutic trails, and to the physicists, without whose help radiation therapy would be still empiric.

CURE VS. PALLIATION

Having established the diagnosis of malignant disease—not always a simple process, and one usually requiring the services of a skilled tissue pathologist†—the problem facing the physician is the selection of the best type of treatment—sometimes

† In this connection it is of interest to note that biopsy is only about one hundred years old (Donaldson, F. *Am. J. M. Sc.*, 1853), while practical radiation therapy is perhaps fifty years of age.

* From the Department of Radiology, Stanford University School of Medicine, San Francisco, California, San Francisco Hospital Service. Presented at the Thirty-first Annual Meeting, American Radium Society, Atlantic City, N. J., June 5–7, 1949.

defined as the one with least physical, psychic and economic trauma. The primary decision must be whether treatment should be radical (to cure), palliative (to alleviate) or merely supportive (to render an advanced process less intolerable).

If cure appears reasonably possible, then shall it be by means of surgery, irradiation or a combination of the two methods? That surgery can cure cancer is accepted by all: that radiology does likewise is still questioned by some. The reasons for this are multiple—psychologic, pedagogic and hereditary! However, for those who will pause to review the numerous microscopically proved ten and twenty year cures achieved by the irradiation method there is no ground for doubt. An adequate dose of ionizing radiation will cure a squamous cell cancer of reasonable proportions, if delivered with proper technique in a specified period of time, just as surely as radical excision or other annihilative methods.[1,5,6]

CHOICE OF METHODS

The choice of surgery or irradiation as the primary method of treatment depends on many factors such as the type and location of the tumor, the condition of the patient, the availability of a competent surgeon or radiologist, and so forth. The type and location of the tumor is of considerable importance and may settle the question alone. For example, a squamous cell carcinoma, well differentiated and of reasonable size, which arises in the rectum of a patient in good general condition, is a purely surgical problem. A small squamous cell cancer of the eyelid is a radiological problem (i.e. best treated by irradiation). A localized cancer of the anterior third of the tongue may be well treated by either method.

Since surgery is the method that has long established itself, one may reasonably ask why and when is radiation therapy preferable? We believe that irradiation is preferable in the treatment *of suitable lesions* for the following reasons:

1. Better cosmetic results (e.g., in cancer of the skin of the face, especially about the eyes, etc.)
2. Better functional results (e.g., in cancer of the lip, tongue, larynx, etc.)
3. Better clinical results (e.g. in cancer of the cervix uteri, in Hodgkin's disease, lymphosarcoma, medulloblastoma, etc.)
4. Less physical strain on patients in poor general condition (cancer of fundus in a hypertensive, etc.)
5. Less economic strain (hospitalization usually not necessary, etc.)*
6. Less psychic strain (in persons with chirurgophobia, etc.).

The advantages of surgery are too well known to need repetition here; the types of cases which we regard as best suited for surgery are outlined in the following pages.

What of the disadvantages of either method? The first and most serious disadvantage is that complete removal or eradication of the tumor may at times not be achievable, despite initially favorable conditions. The next is a series of well known surgical and radiological complications. Necrosis of the ureters may follow a Wertheim operation, or fibrosis a course of pelvic irradiation. Serious hemorrhage may follow a glossectomy or heavy glossal irradiation. The early and late complications of surgery seem to be less stressed than those of irradiation—for reasons concerning which we as radiologists must be philosophic. However, those of irradiation are not negligible and require constant vigilance for reduction and possible prevention. Radiation ulcers; radiation osteonecrosis and chondronecrosis cannot, unfortunately, always be avoided, if one is to achieve cure of the malignancy. The prolonged time sometimes required for cure by irradiation; the painful nature of some radiation reactions, and the atrophic skin changes ulti-

* That a majority of roentgen-ray and many radium therapeutic procedures can be performed efficiently in a properly equipped office is known to all radiologists, but only to a minority of recent graduates—due perhaps to the prolonged institutionalization involved in modern medical training. That radiologists ever *treat* cases is known only to a minority of the public. The relative convenience and economy of ambulatory care for cancer patients is not an idle consideration and needs emphasis to graduates and "planners" alike.

TABLE I

SURGICAL THERAPY USUALLY INDICATED
(Treatment of Choice)

Accessible Sites:
1. Breast (Stage I)
2. Metastatic cervical nodes
3. Corpus uteri (operable)
4. Salivary glands
5. Thyroid

Inaccessible Sites: (Tumor Operable)
1. Brain and spinal cord
2. Esophagus and stomach
3. Small intestine
4. Colon and rectum
5. Kidney
6. Bladder
7. Prostate
8. Lung, etc.

TABLE II

RADIATION THERAPY USEFUL ADJUNCT TO SURGERY

1. Breast (Stage II), postoperative
2. Thyroid, postoperative
3. Uterus, corpus, preoperative
4. Ovary, postoperative
5. Testis, postoperative
6. Bladder
7. Kidney (bulky adenocarcinoma and Wilms' tumor)
8. Miscellaneous tumors, including postoperative recurrence, incomplete removal, etc. (e.g. breast, brain, skin, etc.)

mately present in some cases are other disadvantages. The first is difficult to avoid; the other two are being gradually obviated in most cases.

As a general guide to the use of one or the other method I would like to offer the following series of four tables. They are based on a personal experience of some twenty years in clinical radiology, plus the knowledge of results obtained by other physicians and surgeons. Incidentally, they represent the policy of the Tumor Board on the Stanford University Service at San Francisco Hospital. The tables are designed to indicate the types of tumor best suited at the present time for

Table I. Surgical therapy

Table II. A combination of surgical and radiation therapy

Table III. Radiation therapy alone, and

Table IV. Tumors for which radiation therapy is rarely or never recommended.

It is to be remembered at all times that *in individual cases, circumstances may require departure from this general plan.* However, we believe that the arrangement is a practical and useful one, and of value in maintaining consistency in one's therapeutic approach.

RESULTS OBTAINABLE

The results obtainable by judicious radical surgery and irradiation are well known. Nevertheless a few examples may be of

TABLE III

RADIATION THERAPY USUALLY INDICATED
(Treatment of Choice)

Accessible Sites:
1. Skin
2. Lip and mouth, including tongue
3. Breast (Stage III)
4. Cervix uteri
5. Anal and urethral orifices

Inaccessible Sites:
1. Embryonal tumors—kidneys, testis, ovary
2. Endotheliomas—Ewing's, etc.
3. Medulloblastoma
4. Cancer of the larynx and nasopharynx—selected cases
5. Cancer inoperable, e.g. lung, esophagus, etc.
6. Cancer metastatic, esp. in bone, skin, etc.
7. Lymphoblastoma (Hodgkin's disease and lymphosarcoma) and leukemia

TABLE IV

RADIATION THERAPY RARELY OR NEVER INDICATED

1. Osteogenic sarcoma
2. Miscellaneous "adult" sarcomas
3. Cancer of the intestines
4. Cancer of the gallbladder
5. Cancer of the liver
6. Cancer of the pancreas
7. Advanced cancer with cachexia
8. Acute leukemia
9. Melanoma

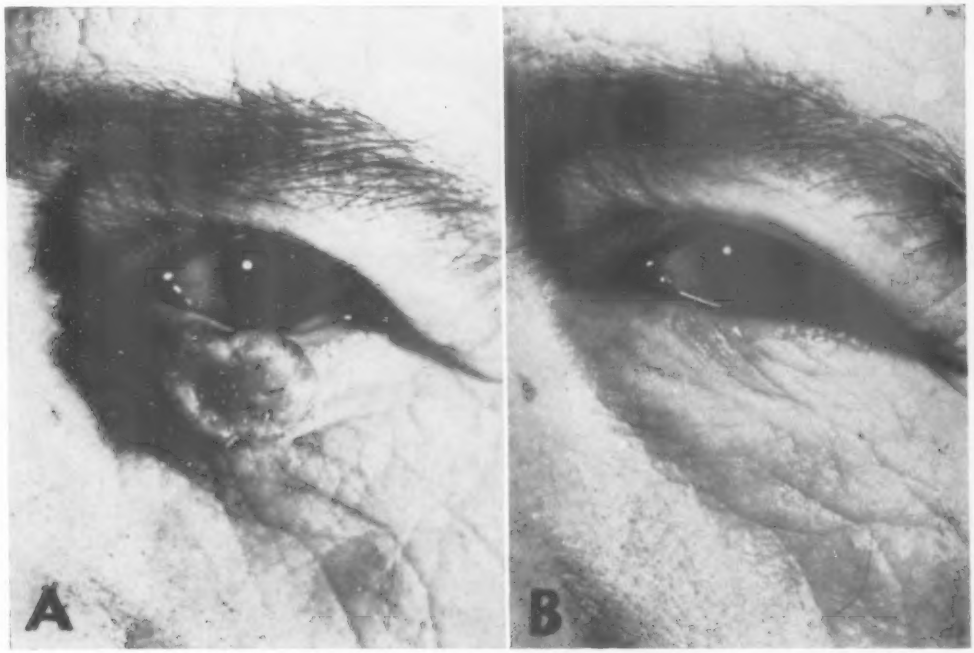

FIG. 1. Case I. Carcinoma of the lower lid. Clinical cure over eleven years. Case before (*A*) and five years after treatment (*B*).

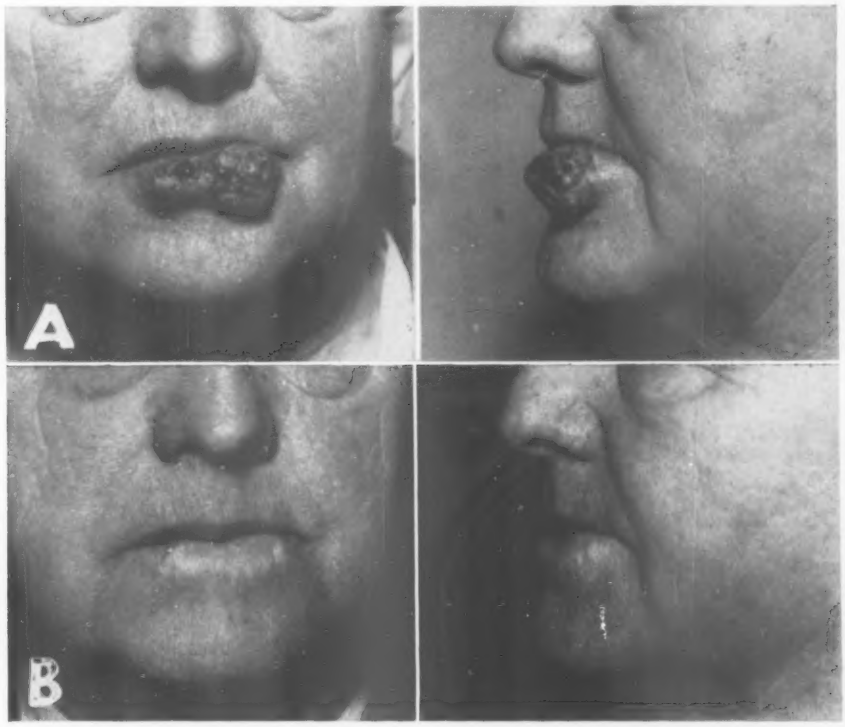

FIG. 2. Case II. Carcinoma of the lip. Clinical cure for nine years. Case before (*A*) and five years after treatment (*B*).

value in illustrating the great benefits of irradiation in some commonly encountered lesions, if only to emphasize the selective place of irradiation.

CASE I. Carcinoma of the lower lid. Male, aged fifty-eight, squamous cell carcinoma, biopsy proved. Treatment: Roentgen irradiation. Tumor dose 6,000 r in fifteen days, to a field 2.5 cm. in diameter (half-value layer 0.3 mm. Cu). Clinical cure over eleven years.

CASE II. Carcinoma of the lip. Male, aged sixty, with squamous cell carcinoma, Grade 3, invading muscle (biopsy proved). Treatment: Roentgen irradiation. Tumor dose 5,000 r in twenty-five days (half-value layer 1.0 mm. Cu) to a field 6 cm. in diameter. Clinical cure nine years.

CASE III. Carcinoma of the larynx. Male, aged fifty-five, with epidermoid cancer, left vocal cord (biopsy proved). Treatment: Roentgen irradiation. Tumor dose 5,500 r in five weeks (half-value layer 1.0 mm. Cu) to a field gradually reduced from 8 to 5 cm. in diameter. Cured ten years.

CASE IV. Carcinoma of the lung. Male, aged fifty-eight, with biopsy verified inoperable bronchogenic carcinoma. Treatment: Roentgen irradiation. Tumor dose approximately 4,000 r in six weeks (half-value layer 1.9 mm. Cu); fields 20 by 15 cm. Relief of cough and hemoptysis: survived two years. Death from cerebral metastases.

CASE V. Hodgkin's disease of the stomach. Male, aged thirty-eight, with known Hodgkin's disease for four years (cervical node biopsy). Ulcerating gastric tumor found following massive oral and rectal hemorrhage (Hgb. 20 per cent; erythrocytes 1.25 million). Treatment: bed rest and roentgen irradiation. Only a low voltage unit was available in his community (half-value layer 0.3 mm. Cu): given 1,000 r to a large epigastric field in three weeks. Lesion reduced in five weeks and not demonstrable in twelve weeks. Patient also had pulmonary and sacral lesions which required subsequent roentgen treatment. Survived nine more years.

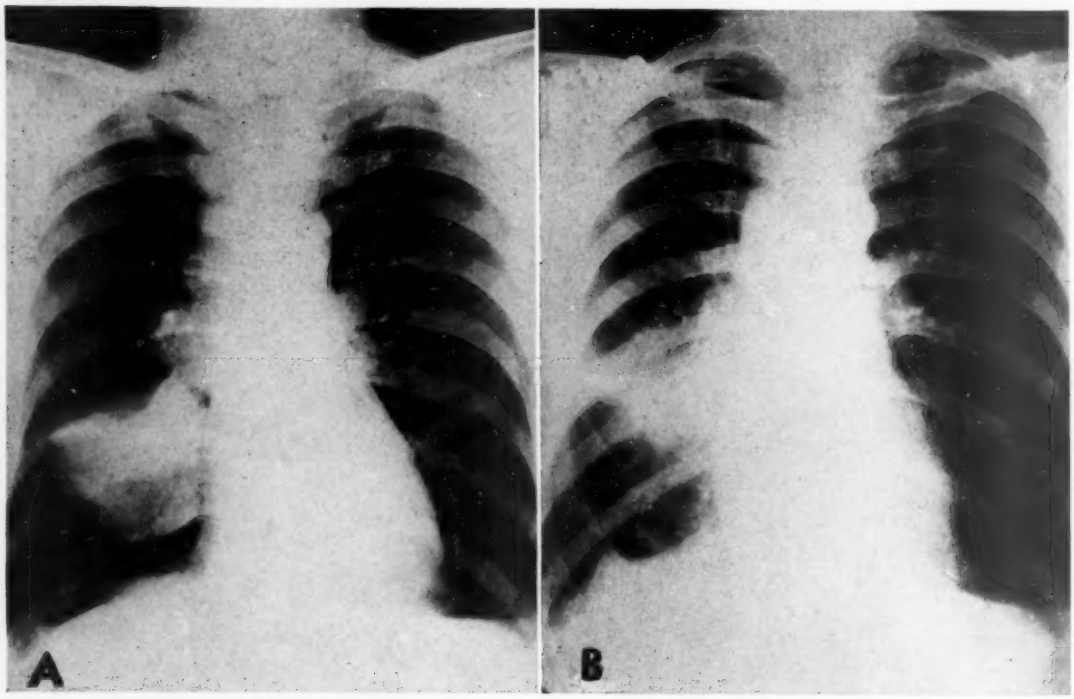

FIG. 3. Case IV. Carcinoma of the lung. Relief of cough and hemoptysis; survived two years. Death from cerebral metastases. Case before (*A*) and one year after treatment (*B*).

CASE VI. Lymphosarcoma of neck, skull and femur. Male, aged thirty-seven, with lympho-

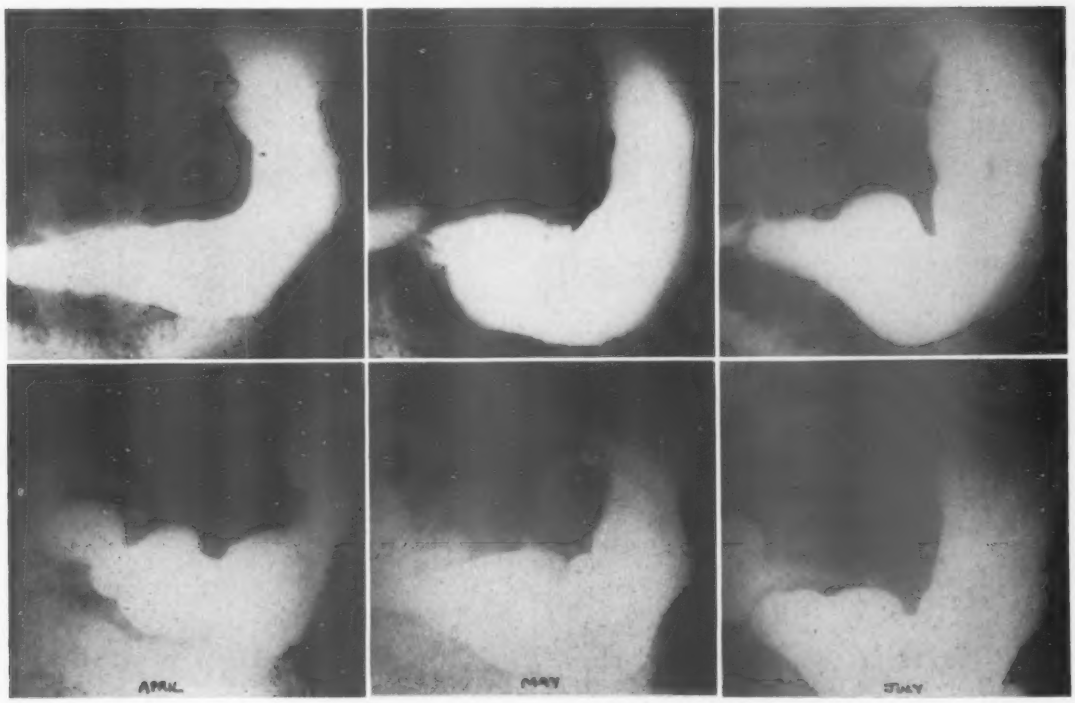

Fig. 4. Case v. Ulcerating gastric Hodgkin's disease, before, during and after roentgen treatment. Tumor dose approximately 500 r in three weeks. Lesion in stomach remained arrested for nine years. Lesions in cervical nodes, lungs and bones also required treatment. Multiple biopsy verification.

sarcoma (cervical node biopsy); severe frontal headaches and pain in left hip for one year. Given "total body" irradiation: 600 r in air, to each side of head, 600 r to entire pelvis and femora, front and back, and 300 r to entire neck and shoulders laterally. Half-value layer 1.0 mm. Cu, 80 cm. distance, fields 40 cm. in diameter. Treatment time only ten days! Gradually improved and returned to work. Roentgen examination one year later showed no lesions. After six years developed left pulmonary and thyroid masses. Given 230 r in air, anterior and posterior, to torso in ten days. Died two years later. Necropsy showed lymphoblastoma, type bizarre (Budd). Survived eight years.

Case vii. Myeloma, plasma cell type, of femur. Female, aged fifty-one, with spontaneous fracture left femur: biopsy of rib showed myeloma. Treatment: splint and roentgen irradiation. Tumor dose 1,000 r in six days (half-value layer 1.0 mm. Cu). Fracture healed. Survived four years and died with meningitis.

In the following tables (v, vi and vii) there are summarized the results obtainable with modern radiation therapy in the hands of such experienced radiologists as Paterson of Manchester, and Hamann of Chicago.

Table V

FIVE YEAR RESULTS OF RADIATION TREATMENT IN 8,298 CASES

(Mx. proved cancer, miscl. sites, Holt Radium Institute, 1946)	
Early cases only (one-third of the total)	86% survival
Late cases only (less than one-half of the total)	13% survival
All cases treated: total percentage alive	40%

In connection with Table vii it is realized that a significant number of the favorable results, especially the five year survivals in these particular diseases, is not necessarily due to radiation therapy. Some cases of leukemia and malignant lymphoma will survive for five years without treatment of any type; such persons have a

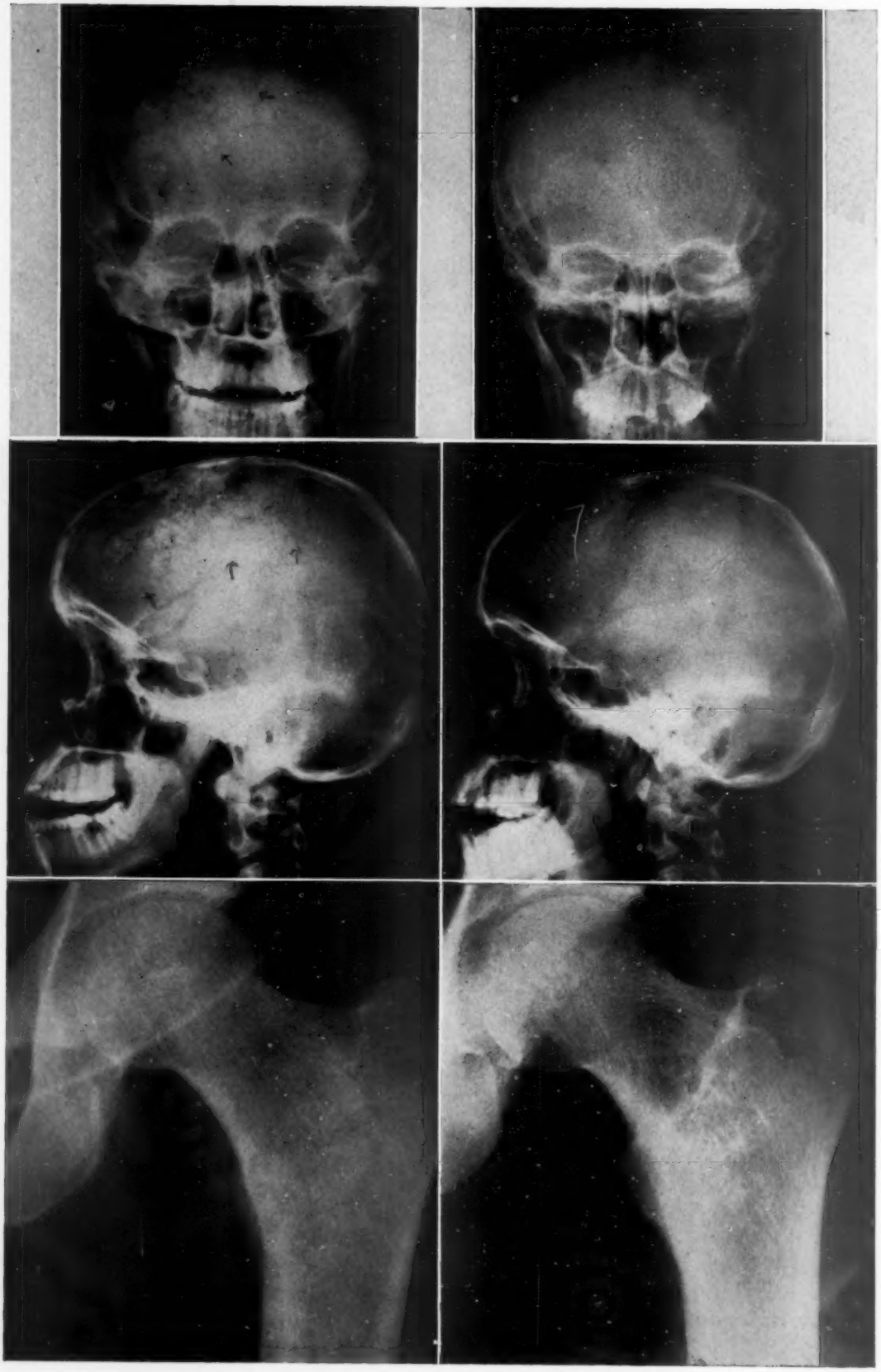

TABLE VI

FIVE YEAR SURVIVALS FOLLOWING RADIATION THERAPY, BY SITES (HOLT RADIUM INSTITUTE)

Cancer of the skin	1803 cases	88%	(98% of 1236 early cases)
Cancer of the breast including post-operative recurrence	863 cases	20%	(55% of 69 early cases)
Cancer of the uterus and cervix	826 cases	28%	(65% of 48 early cases)
Cancer of the lip and mouth	1289 cases	30%	(64% of 300 early cases)
Cancer of the uterus corpus	91 cases	26%	(71% of selected early cases)

relatively mild and chronic form of the disease. The exact percentage of these is difficult to ascertain, and is sufficiently small not to invalidate the statement that judicious irradiation with roentgen rays greatly increases the effective life and comfort of most patients, and prolongs the life of a few. Irradiation may be a life-saving procedure in patients with obstructive mediastinal and diffuse infiltrating renal lymphomas.

CURABLE CANCERS

In the foregoing paragraphs and tables we have referred to cancers as being of two general types, accessible and inaccessible. The former appear to be curable in a good percentage of cases; the latter are a source of less fruitful outcome. What are the apparent *facts* today as to the curability of cancer in general? We believe that the following data, compiled from many sources, give a clue as to the answer.

The incidence of cancer according to site in living persons in the United States is estimated to be approximately as follows.[1,3,4] The figures pertain to the combined sexes, and for purposes of brevity we list only the ten most common sites:

1. Breast
2. Skin
3. Stomach
4. Uterus (cervix:corpus::4:1)
5. Colon
6. Rectum and rectosigmoid
7. Prostate
8. Bladder (and urinary system)
9. Lung (and respiratory system)
10. Lip (and buccal cavity)

The *mortality of cancer*, according to available statistics is as follows.[4,10] (It is fully realized that the accuracy of mortality data is not of high order; nevertheless it is believed that the census reports and published autopsy figures give a guide to the general picture.) Mortality by sites

TABLE VII

RESULTS OF RADIATION THERAPY IN LYMPHOMAS (HAMANN)
(Survival *after* beginning of roentgen therapy)

Type	Cases	3 yr.	5 yr.
Chronic myelogenous leukemia	49	33%	14%
Chronic lymphogenous leukemia	51	30%	23%
Hodgkin's disease	143	35%	21%
Lymphosarcoma	77	12%	9%

(Average survival in first three groups approximately 32 mo.) (*Radiology*, 1948, *50*, 376)

in order of frequency—8 most common sites:

1. Stomach
2. Intestines (excluding rectum)
3. Breast
4. Uterus
5. Liver
6. Lung
7. Male genitals (including prostate)
8. Rectum

The *curability of cancer*. According to sources such as the Registry of Cases of

FIG. 5. Case VI. Lymphosarcoma of skull and left femur (lesser trochanter). Patient also had cervical node involvement (biopsy proved) and, presumably, other deep-seated lesions. He was therefore treated by roentgen irradiation to the skull and torso, including the femora, 600 r(air) to entire area in ten days (see text for details). Survived eight years, ultimately succumbing to his disease.

FIG. 6. Case VII. Plasma cell myeloma of femur, before (*A*) and after (*B*) roentgen treatment, showing healing of the pathological fracture. Tumor dose 1,000 r in six days. Survived four years. Death from independent causes. (Courtesy of *Radiology*.)

Cured Cancer of the American College of Surgeons, this is as follows.[2,5,7] Six most often cured lesions:

1. Breast
2. Uterus (cervix)
3. Lip and mouth
4. Colon and rectum
5. Skin
6. Uterus (fundus)

From these data, and from the experience of our own Tumor Service and practice during the last twenty years, we believe that the 6 most commonly encountered curable cancers are those of the

1. Skin
2. Breast
3. Uterus (cervix)
4. Colon and rectum
5. Lip and mouth
6. Uterus (fundus)

INDICATED METHODS OF TREATMENT

With the realization that cure is possible in a high percentage of early cases of accessible cancer, what then are the facts as to the relative roles of surgery, irradiation and other methods in the treatment of these cases? Making due allowance for the *stage* of disease often present when our patients first consult or reach us, we believe that the following is a fair estimate of the need and indications for the different methods of treatment in the six most often curable groups (Table VIII).

Table VIII indicates that radical or palliative surgery alone is indicated in about 33 per cent of the theoretical group of cases; radical or palliative irradiation in 43 per cent; surgery plus irradiation in 17 per cent; and no surgery or irradiation in about 7 per cent. Of the breast cases especially, a certain number, at least one-third, will need irradiation or other therapy for metastases developing some years after radical surgery.

Now, of course, no one surgeon, radi-

Table VIII

ESTIMATED PERCENTAGE OF CASES SUITABLE FOR INITIAL TREATMENT BY FOLLOWING METHODS:

Tumor[1] Site	Radical Surgery Alone	Radical Surgery plus Irradiation	Radical Irradiation Alone[2]	Palliative Surgery	Palliative Irradiation	Medical Only[3]
Skin	25%	0%	65%	5%	5%	0%
Breast	40	30	10	5	5	10
Uterus (cervix)	5	0	60	5	25	5
Colon and rectum	60	0	0	30	0	10
Lip and mouth	15	20	55	0	5	5
Uterus (fundus)	10	50	20	0	10	10

[1] This table lists only the first six groups of commonly curable cancers.
[2] Note that this column is high because skin, cervix and lip cases predominate.
[3] These are mostly very advanced cases, not offering much hope of palliation.

ologist or clinic will see an equal proportion of all six types of case listed in Table VIII. Indeed the *relative* frequency of these six groups is approximately as follows (according to Levin, Paterson *et al.*):

Breast	8	Colon and rectum	4.5
Skin	5	Lip and mouth	1
Uterus (cervix)	4	Uterus (fundus)	1

If the figures in Table VIII are corrected for this relative incidence, we find the results shown in Table IX.

Table IX

ESTIMATED PERCENTAGE OF CASES SUITABLE FOR INITIAL TREATMENT BY VARIOUS METHODS, CORRECTED FOR RELATIVE FREQUENCY OF DIFFERENT TYPES OF CANCER

a. Radical surgery	32%
b. Radical surgery and irradiation	13%
c. Radical irradiation	31%
d. Palliative surgery	9%
e. Palliative irradiation	8%
f. Medical only	7%
	100%

It therefore seems quite evident that radiation therapy is at least of equal importance with surgery in the best care of the vast majority of cancers curable at the present time.

This fact, that irradiation is of equal importance to surgery, in the cure of accessible cancers, indicates the need for a reorientation of attitude towards Tumor Services and allied endeavors. A medical group or clinic which lists a dozen surgeons and but one radiologist on its staff is not unusual at the present time. Can this be regarded as modern, from the point of view of providing a good cancer therapeutic service?

Joint treatment: In cases which require a combination of surgical and radiological procedures, it is most desirable, if not essential, that *consultation* be had *prior* to the initiation of treatment. In this manner, the place and timing of the different techniques can be planned with maximum benefit to the patient. One of the major and recurring problems in all fields is that "what is familiar to one may not be at all familiar to others." The Consultative Tumor Board, with follow up service, is a valuable method of keeping practitioners informed as to the potentialities of the various cancer therapeutic disciplines. This is especially so since the general practitioner is reported to see, on the average, only 4 new cases of cancer per annum.

RELATIVE MERITS OF DIFFERENT IRRADIATION TECHNIQUES

The best technique is that which will deliver the minimum effective lethal dose to the involved area, with the least injury to the surrounding tissues. Since there is no discernible difference in biological effect between different qualities of radiation from 50 kv. to 1,000 kv., provided tissue

dose and rate of administration are identical, the source of radiation is obviously not of critical importance. One may cure accessible cancers with 60 kv., 100 kv., or 500 kv. apparatus, provided the physical properties of the beam are fully appreciated and are correctly employed. Some years ago Truesdale[8] observed that: "In the application of surgical treatment for cancer, the excellence of instruments is of little value unless the surgeon is cancer-minded in their use." A similar principle applies to irradiation.

In this connection it may be observed that just as surgery is best performed by a surgeon, so, I believe, is radiation best applied by a radiologist. One cannot perform radical surgery or radical irradiation by prescription, nor does a six months course in either discipline always make one a joint specialist! Sound cancer care would seem to require the availability and cooperation of both surgeons and radiologists.

Radium is essential for the care and cure of most cases of cancer of the cervix uteri, many cases of cancer of the uterus corpus, lingual cancer, maxillary antrum tumors and cancer in a few other sites. Ordinary, flexible 200 kv. roentgen-ray apparatus provides the main roentgen weapon for efficient assault upon suitable cases of intraoral, laryngeal and mammary carcinoma, as well as the treatment of the large group of tumors of many other sites suitable only for palliation.

Supervoltage techniques have occasional advantage in the treatment of certain inaccessible, deep-seated tumors. Beams from betatrons and synchrotrons are still of incompletely explored usefulness. Radiations from unstable isotopes are invaluable in research, but of very limited value in cancer therapy. Irrespective of the radiation equipment available, it is the training and experience of the radiologist which is of primary importance: *he* is the "facility"! At the risk of being tautologic, I would repeat these words: a knife never cured a cancer—a *surgeon* using a knife cured it; conversely a roentgen-ray beam never cured—a radiologist using it did.

PALLIATIVE TREATMENT

Just as many types of cardiovascular and renal disease are incurable when first discovered or brought to medical attention, so are approximately 50 per cent of cancers at the present time. The problem of palliation is therefore great. Judicious surgery and irradiation are again our principal weapons —chemotherapy and other aids being of secondary use, except in a few genital tract tumors.

It is natural that the failure which occurs with any one method in a certain proportion of cases, should cause patients and physicians to try out every new "discovery." However, such excursions should be tempered with discretion and preferably preceded by valid clinical tests. Take, for example, the recent enthusiasm for steroids in the treatment of bone metastases from breast cancer. The yield, in terms of pain relief and temporary healing of the lesions, is about 45 per cent with steroids (testosterone, stilbestrol, etc.), while with properly applied roentgen irradiation it is fully 70 per cent.

RELATIVE YIELDS IN CANCER WORK

The amount of energy devoted to the attempted control of gastric cancer is now very great. Experiments in mass survey roentgen methods are afoot: persons with certain types of anemia and hypochlorhydria are being examined periodically: gastric ulcers are being resected with increasing frequency, and total gastrectomy is a common operation. Despite all this activity the five year absolute cure rate is still not much above *3 per cent.*

On the other hand, the control of uterine cervical cancer (which is nearly as common as stomach cancer—there being about 75 cervix cases for every 100 gastric cancers) arouses only moderate interest. It is essentially a radiological disease, and at least *40 per cent* of all cases properly treated survive for five years. Is it not

important to stress the great salvage obtainable in this type of cancer?

SUMMARY

The roles of surgery and irradiation in the cure and care of cancer are complementary and not competitive. The most certain cure for cancer is complete surgical removal or complete destruction by irradiation.

Modern radiation therapy, like modern surgery, is greatly improved in performance, thanks to increasing experience, better follow-up and the use of modern biological adjuncts. If the commonly curable cancers are considered—those of the skin, oral cavity, breast, uterus and rectum—the relative indications for the two methods are virtually equal. That is, while surgery is the curative method of choice in most breast and almost all rectal and rectosigmoid lesions, irradiation is indicated in most cutaneous, oral and uterine ones.

The cure rate in early accessible cancer is encouragingly high—from 65 per cent to 95 per cent of treated cases, depending on the site and type of tumor. The absolute five year clinical cure rate, all cases and stages considered, is estimated to be about 40 per cent in this accessible group.

Since approximately one-fourth of the cancers in males, and two-thirds of those in female patients are accessible, the magnitude of the problem, and the degree of improvement attainable with our present day knowledge may be readily visualized.

The role of radiology being of equal significance to that of surgery in the care and cure of these accessible tumors, it would appear sensible to suggest that the radiological staff have comparable representation and responsibility on tumor policy boards, and be numerically large enough to fulfill their part in the staffing of offices, hospitals and clinics which accept cancer patients for treatment.

The cure rate in inaccessible cancer is still low: tumors of the stomach, colon, prostate or lung give a five year absolute clinical cure rate of less than 10 per cent. Since this group of tumors is roughly as large as the accessible group, the challenge is indeed great. The scalpel is our main hope of cure in early cases of these lesions; and the scalpel or ionizing beam our main hope of palliation in the remainder. In other words, the skillful, radical surgeon is still supreme in this field.

The surgical treatment of advanced visceral cancer and the chemotherapeutic approach to generalized tumors have captured the attention of many clinicians in recent years—to the depreciation of achievements in connection with the more common tumors. Sound thinking suggests that we now devote increasing attention to the curable cancers which can, by simple physical examination, be detected early, and by simple extirpation or irradiation often cured. Proper heed to this group should double our present over-all cure rate.

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AN ANALYSIS OF THE PHYSICAL FACTORS CONTROLLING THE DIAGNOSTIC QUALITY OF ROENTGEN IMAGES*

PART V. UNSHARPNESS

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I. INTRODUCTION

MOST roentgenologists are familiar with the loss of detail which occurs in a roentgenogram when the anatomical structure under examination moves during exposure or when the target size of the roentgen tube is excessively large. This effect is caused by the diminished sharpness with which roentgen images are recorded under these circumstances.

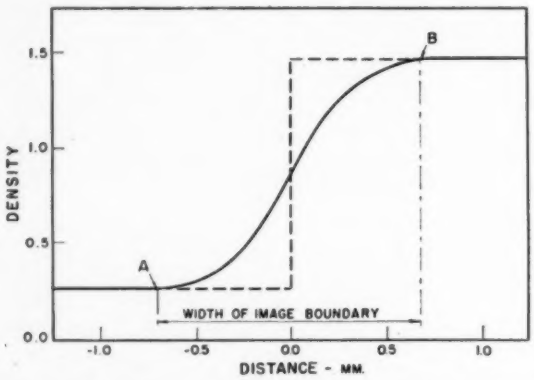

FIG. 1. Manner in which the density of a typical roentgenographic film changes as one progresses across an image boundary. Dotted lines indicate the form which would be taken by the curve if the image were recorded with perfect sharpness.

In addition to target size and anatomical movement, sharpness is also affected by inherent characteristics (e.g., graininess) of roentgenographic films and intensifying screens. Thus, the roentgenographic image of a carefully machined lead plate, when examined closely, will not present an entirely sharp border; instead, the transition in density across the image boundary will be gradual due to the finite size of the grains in the photosensitive emulsion and in the fluorescent screens.

The sharpness of a roentgenographic image may be determined quantitatively by placing the film in a microdensitometer and measuring the density of the film as the image boundary is moved over the entrance slot of the instrument. A typical curve in which film density is plotted as a function of distance is shown in Figure 1. If the image were recorded with perfect sharpness, the relationship between density and distance would have been as shown in the dotted curve; i.e., an abrupt change in density would have occurred when the image boundary passed through the microdensitometer. Since no roentgenographic image is recorded with perfect sharpness, the transition in density is more gradual, like that shown by the solid curve.

The distance between the upper and lower points of inflection in the microdensitometric curve (points *A* and *B* in Fig. 1) constitutes the width of the image boundary. In general, images in which this distance is large appear less sharp than those in which the distance is small. *Accordingly, the width of the image boundary is frequently referred to as the unsharpness of the image.*

The interpretability of a roentgenographic image is profoundly influenced by its unsharpness. Images having a low level of unsharpness are perceived with excellent clarity, whereas those possessing a high level of unsharpness are seen poorly. It will therefore be well to examine carefully the various factors which contribute to roentgenographic unsharpness so that exposure conditions may always be chosen in which their effect is small.

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II. THEORY OF UNSHARPNESS

The three principal factors which influence roentgenographic unsharpness include (1) the finite size of the target of the roentgen tube, (2) the movement of the anatomical part under examination and (3) the inherent characteristics of roentgenographic films and screens. Under most circumstances, two or more of these factors operate collectively to cause image unsharpness. However, before an understanding may be gained of the manner in which these factors operate together it will be necessary to consider each of them individually.

a. Target Size. The effect of target size on image unsharpness is clearly demonstrated by the schematic diagram shown in Figure 2 where F is the target of a roentgen tube, O is a partially radiolucent and motionless object and R is a roentgenographic film. It is apparent that all portions of the film to the left of the point x_0 receive a uniform exposure. Progressing to the right from x_0, the film receives a gradually increasing exposure as the target becomes uncovered, until the point x_1 is reached; beyond this point the film receives a uniform exposure. If the target is emitting uniformly over its surface, the intensity of the radiation reaching any point of the film between x_0 and x_1 is given by the equation

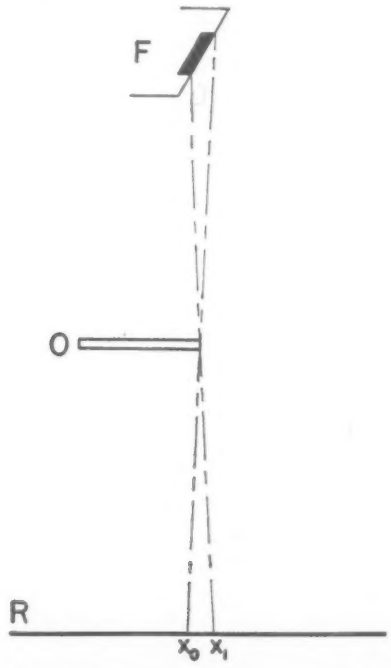

Fig. 2. Diagram illustrating the manner in which the finite size of the target of the roentgen tube causes unsharpness. F, target of roentgen tube; D, partially radiolucent object; R, roentgenographic film.

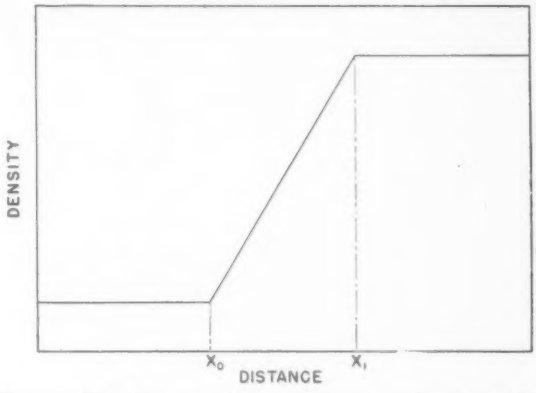

Fig. 3. Density vs. distance curve produced at the image boundary of the film exposed in Figure 2.

$$G_x=\frac{(G_{x_1}-G_{x_0})(x-x_0)}{(x_1-x_0)}+G_{x_0} \qquad (1)$$

where G_{x_1} is the intensity at and to the right of point x_1

G_{x_0} is the intensity at and to the left of point x_0 and

x is the position of any point between x_0 and x_1.

Since the exposure received by a film is equal to the product of the roentgen intensity by exposure time, the exposure received by any point of the film between x_0 and x_1 is therefore

$$E_x=\frac{(G_{x_1}-G_{x_0})(x-x_0)t_a}{(x_1-x_0)}+G_{x_0}t_a \qquad (2)$$

Where t_a is the exposure time. Now the roentgenographic density produced in an exposed and processed film is essentially proportional to the exposure (See Fig. 4, part IV*). Therefore, the density at any point of the film between x_0 and x_1 is

$$D_x=\frac{(G_{x_1}-G_{x_0})(x-x_0)kt_a}{(x_1-x_0)}+G_{x_0}kt_a-c \qquad (3)$$

* Am. J. Roentgenol. & Rad. Therapy, 1946, *55*, 627–633.

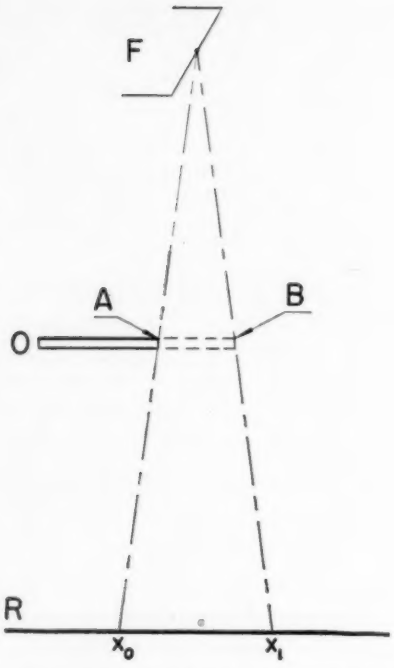

FIG. 4. Diagram illustrating the manner in which the movement of an object being roentgenographed causes unsharpness. *F*, dimensionless roentgen tube target; *O*, partially radiolucent object; *R*, roentgenographic film.

where k and c are constants.

It is evident from equation (3) that as one progresses from x_0 to x_1, density increases linearly with distance if the roentgenographic film has no inherent unsharpness of its own. The microdensitometric curve at the image boundary will therefore have the form illustrated in Figure 3. The distance between the upper and lower points of inflection of this curve is clearly x_1-x_0. Therefore the geometric unsharpness produced by the finite size of the target of the roentgen tube is given by the equation

$$U_g=x_1-x_0=\frac{Fm}{d-m} \tag{4}$$

where F is the target size, m is the object-film distance and d is the target-film distance.

b. Anatomical Movement. The manner in which anatomical movement, alone, may produce image unsharpness is demonstrated schematically in Figure 4, where F is the dimensionless target of a roentgen tube, O is a partially radiolucent object whose edge moves from position A to position B, during exposure and R is a roentgenographic film. It is clear that all portions of the film to the left of the point, x_0 receive a uniform exposure. To the right of x_0, the film receives an additional exposure, the amount of which depends on how long that portion of the film remains uncovered during the exposure.

If the object moves uniformly across the film, the intensity of the radiation reaching any point of the film to the right of the point x_0, and to the left of the point x_1 (the position of the projected edge of the object at the end of the exposure), is given by the equation

$$G_x=\frac{(G_{x_1}-G_{x_0})(x-x_0)}{(x_1-x_0)}+G_{x_0} \tag{5}$$

where G_{x_1} and G_{x_0} have the same significance as in equation (1). Now equation (5) has the same form as equation (1). Therefore, equations (2) and (3) apply also to the condition where a uniformly moving object passes over a roentgenographic film during exposure. Accordingly, the microdensitometric curve at the image boundary will have the form given in Figure 3 and the unsharpness value produced by the moving object will be x_1-x_0; that is,

$$U_m=x_1-x_0\equiv vt_a=\frac{v_0t_ad}{d-m} \tag{6}$$

where v_0 is the velocity of the object O and v is the velocity of projected edge of the object's shadow on the film.

The foregoing derivation assumes that the object being roentgenographed moves uniformly during exposure. In practice, however, this situation seldom occurs. Instead, the movement is likely to be quite complex; furthermore, the degree of complexity is likely to vary considerably from one examination to another. An analysis of such movements therefore is most difficult and it is necessary to resort to the above derivation as a first approximation to actual roentgenographic conditions.

c. Inherent Unsharpness. As stated in earlier paragraphs, the roentgenographic image of a motionless object, exposed with an effectively dimensionless roentgen tube target is never perfectly sharp but instead produces a microdensitometric tracing similar to that illustrated in Figure 1. Unlike the curve shown in Figure 3, this curve is sigmoid in shape, a characteristic which will have considerable interest in later paragraphs.

d. Effect of Several Unsharpness Factors Operating Simultaneously. Equation (3) specifies the unsharpness conditions when a stationary object is roentgenographed with a target of finite size or when a moving object is exposed with a dimensionless roentgen tube target. When target unsharpness and movement unsharpness occur simultaneously, the relationship is no longer valid.

Figure 5 illustrates schematically the prevailing conditions during the roentgenography of a moving object by a roentgen tube having a target of finite size. As in Figure 2, F represents the target of the roentgen tube, O is a partially radiolucent object and R is the roentgen film. During the exposure the object moves, let us assume uniformly, from A to B. From the diagram it is evident that the intensity of the radiation reaching the film at all points to the left of x_0 is uniform. When the object is at position A, the intensity of the radiation at any point on the film between points x_0 and x_1 is that given in equation (1). However, as the object moves from position A to position A_1, the intensity of the radiation at points between x_0 and x_1 gradually diminishes with time and the exposure received by any portion of the film within these limits is given by the function

$$E_x = \int_0^{t_a} G_{tx}\,dt \qquad (7)$$

where G_{tx} is the intensity of the radiation falling on any point, x, of the film between x_0 and x_1 at the instant, t, and t_a is the exposure time of the film. Now, from equation (1) and an inspection of Figure 5, it is evident that the value of G_{tx} is simply

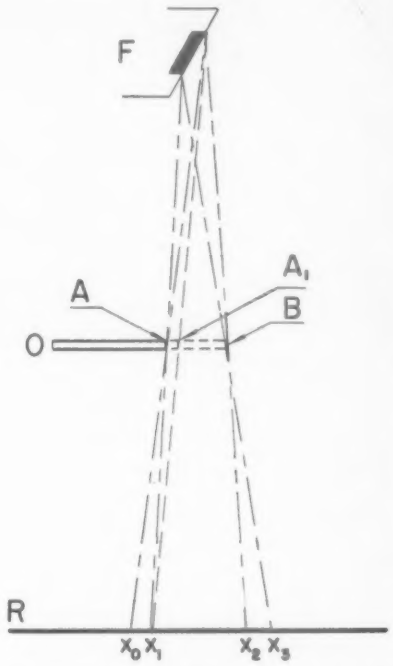

Fig. 5. Diagram illustrating the manner in which the finite size of the target of the roentgen tube and movement of the object being roentgenographed operate collectively to cause unsharpness. Symbols are the same as in Figures 2 and 4.

$$G_{tx} = \frac{(G_{x_1} - G_{x_0})(x - x_0 - vt)}{(x_1 - x_0)} + G_{x_0} \qquad (8)$$

for values of

$$t \leqq \frac{x - x_0}{v}$$

and

$$G_{tx} = G_{x_0} \qquad (8a)$$

for values of

$$t > \frac{x - x_0}{v}$$

where G_{x_1} and G_{x_0} have the same significance as in equation (1) and where v is the velocity with which the image boundary of the object, O, travels across the film; that is,

$$v = \frac{x_1 - x_0}{t_1} \qquad (9)$$

where t_1 is the time needed for the object O, to travel from A to A_1. When equations (8) and (8a), within their respective limits, are substituted in equation (7)

$$E_x = \int_0^{t_x} \frac{(G_{x_1} - G_{x_0})(x - x_0 - vt)}{(x_1 - x_0)} \cdot dt + \int_0^{t_a} G_{x_0} \cdot dt \tag{10}$$

where

$$t_x = \frac{x - x_0}{v}.$$

Therefore when the integration is completed, it will be seen that the exposure received by any point, x, of the film between x_1 and x_0 is

$$E_x = \frac{(G_{x_1} - G_{x_0})(x - x_0)^2 t_1}{2(x_1 - x_0)^2} + G_{x_0} t_a \tag{11}$$

where t_a is the total exposure time of the film. Since the relationship between roentgenographic density and exposure is essentially linear, the density at any point x, between x_0 and x_1 is

$$D_x = \frac{(G_{x_1} - G_{x_0})(x - x_0)^2 k t_1}{2(x_1 - x_0)^2} + G_{x_0} k t_a - c \tag{12}$$

where k and c are constants. It will be observed that within the region between x_0 and x_1, density varies as the *square of the distance* across the image boundary.

The density within the image boundary region between x_1 and x_2 may be readily determined by considering the time of exposure within this region as consisting of three phases: (1) the time during the initial stages of the exposure when the film receives radiation directly from the roentgen tube; (2) the time during the middle stages of the exposure when the border of the moving object and the finite target size combine to project on the field a penumbral shadow and (3) the time during the late stages of the exposure when the film receives radiation of uniform intensity through the body of the moving object.

In the first stage the exposure received by the film at some position, x, between x_1 and x_2 (Fig. 5) is given by the equation

$$E_{xa} = G_{x_1} t' \tag{13}$$

where G_{x_1} has the same significance as formerly and t' is the time required for the object, O, to move from its starting position, A, to a position at which its border begins to produce a penumbral shadow of the target at the point, x. This time, of course, is a function of the velocity of the object and is given by the equation

$$t' = \frac{x - x_1}{v} \tag{14}$$

where v, the velocity, is defined by equation (9). Therefore, equation (13) becomes

$$E_{xa} = G_{x_1} t_1 \cdot \frac{(x - x_1)}{(x_1 - x_0)}. \tag{15}$$

In the second stage of the exposure the intensity of the exposing radiation varies with time as the object covers more and more of the presenting surface of the target from the film. The value of the exposure received by the film therefore may be determined by employing similar methods to those used in deriving equation (11). From equation (8) it is evident that the intensity of the exposing radiation at any instant is given by the equation

$$G_{xb} = \frac{(G_{x_1} - G_{x0})(x_1 - x_0 - vt)}{(x_1 - x_0)} + G_{x_0}. \tag{16}$$

Therefore, the exposure received by the film during this phase of the exposure is

$$E_{xb} = \int_0^{t_1} \frac{(G_{x_1} - G_{x_0})(x_1 - x_0 - vt)}{(x_1 - x_0)} dt + \int_0^{t_1} G_{x_0} dt$$

or

$$E_{xb} = \frac{(G_{x_1} - G_{x_0}) t_1}{2}. \tag{17}$$

During the third phase of the exposure the intensity of the exposing radiation is again constant at a value G_{x_0}. The dura-

tion of this phase is equal to the total length of exposure less the time necessary to complete phases (1) and (2). Therefore, the exposure received by the film during phase 3 is given by the equation

$$E_{xc}=G_{x_0}\left(t_a-\frac{(x-x_0)}{(x_1-x_0)}t_1\right)\cdot \qquad (18)$$

When equations (15), (17), and (18) are added, the total exposure received by any point, x, of the film between x_1 and x_2 is

$$E_x=G_{x_0}t_a+\frac{(2x-x_1-x_0)(G_{x_1}-G_{x_0})t_1}{2(x_1-x_0)}\cdot \qquad (19)$$

The density of the film at this point will therefore be

$$D_x=G_{x_0}kt_a+\frac{(2x-x_1-x_0)(G_{x_1}-G_{x_0})kt_1}{2(x_1-x_0)}-c\cdot (20)$$

Equation (20) may also be written in the form

$$D_x=G_{x_0}kt_a+(2_x-x_1-x_0)(G_{x_1}-G_{x_0})k/2v-c\cdot (20a)$$

It will be observed that within the region between x_1 and x_2 density increases linearly as the image boundary is crossed. Indeed the rate of increase is identical to that which would occur if the only unsharpness present were that due to movement of the object.

An expression giving the density within the region between x_2 and x_3 may be easily derived by employing methods similar to those used in the derivation of equation (11) and takes the form

$$D_x=G_{x_1}kt_a-\frac{(G_{x_1}-G_{x_0})(x_3-x)^2kt_1}{2(x_3-x_2)^2}-c\cdot \qquad (21)$$

Here, as in the region between x_0 and x_1, the density varies as the square of the distance across the image boundary.

In the foregoing analyses, it was assumed that the penumbral shadow of the object remained equal in width as the object moved from A to B. Such an assumption, however, is valid when the target size and object are small in respect to target-object and target-film distances. Also, it was assumed that the unsharpness due to movement of the object was greater than that due to the size of the target. However, if the reverse were true, it could easily be shown that equations (12), (20) and (21) would be equally valid.

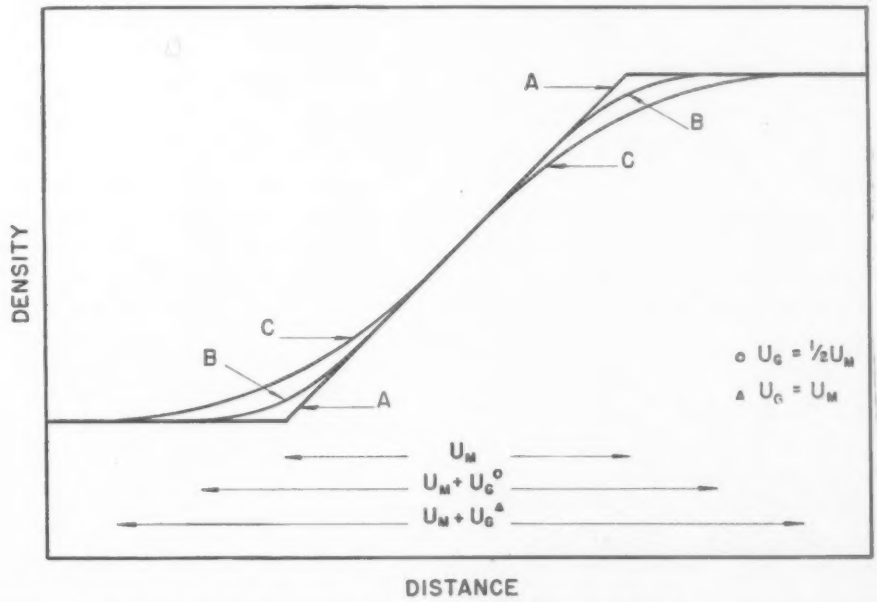

Fig. 6. Density of a film at any point within the image boundary produced by a moving object and roentgenographed with a roentgen tube target of finite size. Curve A, target unsharpness is zero; curve B, target unsharpness is equal to one-half the value of movement unsharpness; curve C, target unsharpness and movement unsharpness are equal.

The density of a film at any point within the image boundary of a moving object roentgenographed with a target of finite size may be determined from equations (12), (20), and (21). In Figure 6, such curves are plotted for three combinations of target size and object movement. In curve *A*, one of the parameters is zero (relatively much smaller than the other); in curve *B*, one of the parameters has an unsharpness equal to one-half of that of the other; whereas in curve *C*, the unsharpnesses are equal. It will be observed that in each case the distance from the point of inflection of the curve below to the point of inflection of the curve above is equal to the sum of the individual unsharpness values (U_m+U_g). Therefore, the *total* unsharpness of an image, where several types of unsharpness are present, is equal to the sum of the individual factors; i.e.

$$U_t = U_1 + U_2 \cdots U_n \qquad (22)$$

where U_t is the total unsharpness. Although equation (22) is mathematically valid, it apparently does not indicate the *observed or effective* unsharpness of the roentgenographic image. This fact was first discovered by Warren[1] several years ago. In his experimental studies, Warren prepared a series of films in which a uniformly moving lead plate was employed as a test object. Exposures were made without intensifying screens and with object movement and target size sufficiently high that the unsharpness contributed by the film was insignificantly low. Target unsharpness and movement unsharpness were varied independently of one another through a wide range. The films were then given to disinterested readers to grade in order of unsharpness. The correlation between the observations and equation (22) was rather poor, equation (22) tending to indicate an unsharpness considerably in excess of the observations. This led Warren to devise a number of formulae by which effective unsharpness could be predicted when two types of unsharpness are present.

One of the most striking observations which may be made from Warren's material is the consistency with which effective unsharpness values seldom differ greatly from the largest of the two individual unsharpness levels (target and movement unsharpness) present in his experimental films. Such a situation would not be surprising if one of the unsharpness values were always considerably greater than the other. The effect was observed, however, even when the two values were almost equal; that is, when two types of unsharpness were present and equal to one another, the effective unsharpness was identical to that which would have occurred if only one type of unsharpness were present.

In an effort to explain Warren's findings, the relationships expressed in equations (3), (12), (20), and (20a) were recently given critical review by the author. From this study several observations were made which seem to indicate that the effective unsharpness value of an image is dependent on something much more fundamental than heretofore has been contemplated. *Indeed, it appears that the effective unsharpness of an image is governed by the maximum slope of the microdensitometric curve obtained at the image boundary.* To illustrate this, let us examine the conditions given by the several equations in question.

When one type of unsharpness (target or movement) is present the maximum slope of the density vs. distance curve at the image boundary is obtained by differentiating equation (3); that is,

$$\left[\frac{dD}{dx}\right]_{\max} = \frac{(G_{x_1} - G_{x_0})kt_a}{x_1 - x_0}. \qquad (23)$$

But

$$(G_{x_1} - G_{x_0})kt_a = D_1 - D_0$$

where D_1 is the density in the background of the film and D_0 is the density within the image of the roentgenographed object. Also, $x_1 - x_0 = U_g = U_m$. Therefore,

$$\left[\frac{dD}{dx}\right]_{\max} = \frac{D_1 - D_0}{U_g} = \frac{D_1 - D_0}{U_m}. \qquad (24)$$

Now if two types of unsharpness are present and the value of one (e.g. movement unsharpness) is greater than the other, the maximum slope of the density vs. distance curve is obtained by differentiating equation (20a); that is,

$$\left[\frac{dD}{dx}\right]_{\max}=\frac{(G_{x_1}-G_{x_0})k}{v}. \tag{25}$$

When v is eliminated from equation (25) by means of equation (6)

$$\left[\frac{dD}{dx}\right]_{\max}=\frac{(G_{x_1}-G_{x_0})kt_a}{U_m}=\frac{D_1-D_0}{U_m}. \tag{26}$$

Thus, it is clear from equation (26) that the maximum slope of the curve is a function only of movement unsharpness, that is, the maximum slope again would be determined by the larger unsharpness level.

Finally, if the two unsharpness levels are made equal an examination of Figures 5 and 6 reveals that the maximum slope of the density versus distance curve within the image boundary may be determined by differentiating equation (12) and setting the value of x equal to x_1 and the value of t_1 equal to t_a; that is

$$\left[\frac{dD}{dx}\right]_{\max}=\frac{(G_{x_1}-G_{x_0})kt_a}{(x_1-x_0)}=\frac{D_1-D_2}{U_m}=\frac{D_1-D_2}{U_g}. \tag{27}$$

Thus, when the two unsharpness levels are equal the maximum slope of the curve is the same as though only a single unsharpness were present.

From the foregoing, there is a compelling similarity in the manner in which the maximum slopes of the image boundary curves and the effective unsharpnesses of roentgen images behave. When two types of unsharpness are present, the combined effect appears to be controlled only by the larger of the two unsharpness levels. From this, one may reasonably postulate that an observer's impression of an image boundary is governed by the maximum slope of the boundary. Such a postulate has been further supported recently by additional mathematical analyses and experimental studies of conditions where three types of unsharpness (target, movement, and inherent) are present.

Before discussing these analyses, it should be recalled that in the derivation of equations (20) and (20a) target unsharpness and movement unsharpness were assumed to be linear functions as shown in Figure 3. However, it will be seen in Figure 1 that inherent unsharpness is not a linear function and therefore the mathematical analysis cannot follow a simple extension of the previous derivations to include a third type of linear unsharpness. Now it will be seen from Figure 1 that the form of the inherent unsharpness curve is similar to that of curve B in Figure 6; that is, inherent unsharpness behaves as though it were the result of *two* linear types of unsharpness operating simultaneously. Accordingly, our analysis must be extended to include four types of linear unsharpness.

By pursuing operational mathematics similar to those followed earlier in this paper it may be shown that when four equal and linear types of unsharpness are present, the maximum slope of the image boundary curve is given by the equation

$$\left[\frac{dD}{dx}\right]_{\max}=\frac{2(D_1-D_2)}{3U} \tag{28}$$

where U is the value of any one of the unsharpness levels.

When one unsharpness level is greater than the others, equation (28) approaches

$$\left[\frac{dD}{dx}\right]_{\max}=\frac{D_1-D_2}{U_{\max}}. \tag{29}$$

Thus, when four linear types of unsharpness are present, the maximum slope of the image boundary curve as determined from equations (28) and (29) is equal to that which would have been produced by the largest factor alone except when all four unsharpnesses are equal; then, the maximum slope is 33 per cent less than that produced by any one of the factors singly.

If the perception of unsharpness is governed by the maximum slope of the density vs. distance curve at an image boundary, one would expect from the foregoing calculations that when target, movement, and film-screen unsharpness are contributing equally to an unsharpness level, effective unsharpness will be somewhat less than 50 per cent greater than that produced by any one of the unsharpness factors alone. Effective unsharpness, of course, should not exhibit the full 50 per cent increase predicted by equation (28) since inherent unsharpness does not behave like two equal linear unsharpnesses operating simultaneously as considered in the development of equation (28) but instead like two linear unsharpnesses in which one has about twice the magnitude of the other. It may easily be shown that this characteristic should reduce the effective unsharpness value several per cent depending on the exact form of the density vs. distance curve of the film-screen combination being used.

Klasens[2] has recently conducted experiments to determine visually the relation between effective unsharpness and the values of three individual unsharpnesses, including film-screen unsharpness, and has found that when the three degrees of unsharpness are equal, effective unsharpness is about 45 per cent greater than that produced by any one of the unsharpness factors alone. Such close agreement between observed and theoretical results seems to give strong support to the foregoing postulate regarding the perception of image boundaries. These workers have also shown that when one unsharpness factor is larger than the remainder, effective unsharpness is identical to that which would have been produced by the largest factor. *It, therefore, is clear that effective unsharpness is not equal to the sum of the individual unsharpness values but instead is seldom greater than the largest of these values.*

This conception of unsharpness has far-reaching implications in many roentgenologic problems. It illustrates, for example, that there are limits to which effective unsharpness may be reduced by decreasing the unsharpness of a single factor. Thus when target size is made smaller, unsharpness becomes smaller until target unsharpness equals film or movement unsharpness, whichever is the larger. *Reducing target size still further effects no change in the appearance of a roentgenogram.* The same limitations also apply to movement unsharpness; that is, there is nothing to be gained by reducing movement unsharpness below the levels of target unsharpness or inherent film or film-screen unsharpness. Indeed it is clear that the objective of the roentgenologist when facing the technical problems of unsharpness in his clinical films should be the maintenance of target unsharpness and movement unsharpness at values equal to or slightly smaller than inherent film or film-screen unsharpness (see Table 1). Effort expended in reducing these values to lower levels is largely wasted as far as practical results are concerned.

Table I

UNSHARPNESS CHARACTERISTICS OF NINE COMMERCIAL BRANDS OF INTENSIFYING SCREENS

Screen	Unsharpness
Patterson Detail	0.17 mm.
Patterson Par Speed	0.30 mm.
Patterson High Speed	0.33 mm.
Eastman Kodak High Definition	0.26 mm.
Eastman Kodak Fine Grain	0.29 mm.
Eastman Ultra Speed	0.32 mm.
Buck Detail	0.23 mm.
Buck Mid Speed	0.25 mm.
Buck Extra Speed	0.28 mm.
Patterson Type "D"	0.48 mm.
U. S. Radium 666-D	0.55 mm.

The concept also affects the method by which the inherent unsharpness of a roentgenographic film or film-screen combination should be measured. In equations (24), (26), (27) and (29), the relationship between unsharpness and the maximum slope of the density vs. distance curve takes the form

$$\left[\frac{dD}{dx}\right]_{\max} = \frac{D_1 - D_0}{U}. \qquad (30)$$

When equation (30) is rearranged

$$U = D_1 - D_0 \Big/ \left[\frac{dD}{dx}\right]_{max}. \qquad (31)$$

But

$$\left[\frac{dD}{dx}\right]_{max} = \frac{D_1 - D_0}{\Delta x} \qquad (32)$$

where Δx is equal to the interval along the distance-axis between the intercepts of a line drawn at the maximum slope of the unsharpness curve (density vs. distance curve) with the upper and lower density levels. Therefore

$$U = \Delta x. \qquad (33)$$

That is, the inherent unsharpness of a roentgenographic film or film-screen combination is equal to the distance between the intercepts of a line drawn at the maximum slope of the unsharpness curve with the upper and lower density levels. Therefore, the inherent unsharpness of a roentgenographic film or film-screen combination should be measured by roentgenographing on the film or film-screen combination under test a straight-edge composed of some relatively opaque material and by making a microdensitometric tracing across the image boundary appearing on the film (see Fig. 7). A line should then be drawn at the maximum slope to the curve and the intercepts of the line with the upper and lower density levels noted (points *A* and *B*, Fig. 7). The desired unsharpness value is the distance between the two points, measured along the distance-axis of the graph.

In Table 1 are listed the inherent unsharpness data of a typical roentgenographic film exposed with nine brands of intensifying screens. Since the granularity of intensifying screens is so much greater than that of roentgenographic emulsions, data for other brands of film are identical to those shown in Table 1. These data were derived by roentgenographing a carefully machined lead plate on each material studied. The films after processing were examined microdensitometrically and unsharpness curves prepared. Unsharpness values were then determined according to the method outlined above.

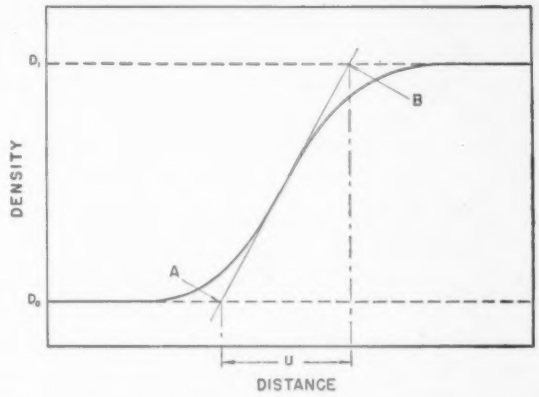

Fig. 7. Graphical illustration of method for measuring the inherent unsharpness of a roentgenographic film. *C*, density vs. distance curve occurring across image boundary appearing in the film; *A*, *B*, line drawn tangentially to maximum slope of curve; *U*, unsharpness value, as given by the difference between the intersepts of the line (*A*, *B*) and the upper and lower density levels.

In all of the above determinations, the films were given sufficient pre-exposure to raise the density of the films beneath the object to a value of 0.8. This density was chosen because it lies near the mid-point of the useful range of film blackening. Accordingly, the data are fairly representative of conditions occurring in roentgenographic practice.

SUMMARY

The images recorded in a roentgenographic film do not appear completely sharp to an observer because of the finite size of the target of the roentgen tube, and the graininess of the film and screens with which the roentgenogram is made. Movement of the objects under examination also contributes to image unsharpness. Until recently it has been thought that when two or more types of unsharpness (target, movement or inherent film-screen unsharpness) occur simultaneously, the overall or effective unsharpness is equal to the sum of the individual unsharpness values. Recent experimental work, however, does not support this belief; indeed, it appears that

when two or more types of unsharpness are present in a film, the effective unsharpness is simply equal to the value of the largest of the several factors except under the condition when all are equal. Then the effective unsharpness value rises slightly.

This concept of unsharpness has far-reaching implications in many roentgenologic problems for it indicates that there are limits to which effective unsharpness may be reduced and the clarity of roentgenographic images improved by decreasing the unsharpness of a single factor. For example, when target size is made smaller, unsharpness becomes smaller until target unsharpness equals film or movement unsharpness, whichever is the larger. Reducing target size still further effects no change in the appearance of the roentgenogram. The same limitations also apply to movement unsharpness. Thus it appears that the roentgenologist, when facing technical problems of unsharpness in his clinical films, should try to maintain target unsharpness and movement unsharpness (factors over which he can exert control) at values equal to or slightly smaller than the inherent unsharpness of his films and screens. Effort expended in reducing these values to lower levels is largely wasted as far as practical benefits are concerned.

Inherent unsharpness values of several roentgenographic films and film-screen combinations are tabulated in the paper.

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THE EXTENDED TUBE TECHNIQUE IN INTRA-ORAL ROENTGENOGRAPHY

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MOST intra-oral roentgenograms are very inadequate, since they fail to exemplify conditions as they actually exist in the mouth. Although it is true that most dental offices today have roentgen-ray machines, as many different types of results are obtained from them as there are dentists using the machines. There are two main reasons for this condition:

(1) The technique of exposing dental films has in the past been a mixture of the knowledge obtained in school and the technique as taught by dental supply salesmen. The end result of such haphazardly learned methods is the production of more inaccurate roentgenograms than true ones.

(2) The average dental machine has a focal distance of approximately 8 inches. With such a focal distance the rays emitted are not parallel but diverge beyond the focal point producing a distorted image upon the film. If we are to render a service to our patients which portrays the conditions as they actually exist, then we must use a technique which will produce those results. Since roentgenograms are one of the main links in the chain of oral diagnosis it is of utmost importance that nothing but the best possible films be used. How many dentists would be willing to measure the image of a tooth produced upon a roentgen film and the actual extracted tooth, and expect to obtain the same measurement? In most instances there would be either a foreshortening or an elongation of the image upon the film. Most of us are attempting to make interpretations of such roentgenograms as this. It must be agreed that most intra-oral roentgenograms are very inadequate.

Dr. Gordon Fitzgerald, Associate Professor of Oral Roentgenology, University of California, has developed a technique whereby any dentist, with any type of a dental roentgen-ray machine, can reproduce upon a roentgen film an image of conditions as they actually exist in the mouth, and do it repeatedly. In his work there is nothing particularly new, for physicians have for years been taking roentgenograms of various parts of the anatomy with target distances of from 30 to 40 inches. They have known that the greater the target or focal distance from the object the more nearly parallel the rays and hence the less distortion of the image. Through his experiments Fitzgerald has found that a maximum distance of 20 inches in intra-oral work produces a true image upon the film and that greater distances have little bearing upon the end result. There are three other factors in this technique which differ from the standard procedures as taught in most dental schools:

(1) The plane of the dental film is always parallel to the long axis of the teeth being roentgenographed. *No* part of the film is *ever* bent.

(2) The film is placed in the mouth *away* from the teeth being roentgenographed. In most techniques the film is placed flat against the tissue forming an angle with the long axis of the teeth and a line bisecting this angle must then be penetrated perpendicularly by the rays.

(3) No definite angulation in the vertical is established for any exposure since all mouths are different.

With these thoughts in mind, it becomes a geometrical problem of placing the patient's head in the proper position, the film in the mouth in the proper position, and the tube of the roentgen machine in the proper relation to the plane of the film both vertically and horizontally. Since the

film is not placed flat against the tissue and should never be bent, it becomes evident that the use of the regular sized dental film ($1\frac{1}{4}$ by $1\frac{5}{8}$ inches) could not be used satisfactorily in the anterior part of the mouth. A narrowed film ($\frac{3}{4}$ by $1\frac{5}{8}$ inches) was developed for this region.

Central Lateral Incisor Region. In the upper arch, such a roentgenogram should portray the following:

(1) Central and lateral incisors centered upon the film, with the contacts open (unless the teeth are excessively overlapped).

(2) The mesial contact of the adjacent central incisor.

(3) Approximately the mesial third of the cuspid.

(4) The floor of the nasal passage.

(5) The median suture line.

(6) The incisive foramen and fossae.

To obtain images of these anatomical structures upon the film, two cotton rolls are placed side by side on the tube surface of the film packet. The patient's thumb supports the tissue edge of the film against the palate. A hinge-like freedom of movement is obtained and the springy characteristics of the cotton rolls allows the packet to be maintained out away from the teeth and parallel to their long axes. The patient bites the film gently to stabilize this retention. The inclinations of the long axes of the central and lateral incisors are the guides in film positioning, unless one or the other of the teeth is malpositioned in the arch. In that case the tooth with the more typical alinement is used. With the two cotton rolls between the film and the lingual surface of the crowns of the teeth and the tissues, the upper edge will be well back in the palate. The vertical angulation is directed so that the rays pass through the two parallel planes: i.e. the plane of the film and that of the long axis of the tooth. The horizontal angulation is so directed that the rays pass through the contact between the central and lateral incisors, or at right angles to the ridge if there is excessive overlapping or if the area is edentulous.

In order to determine the correct positioning of the roentgen tube in relation to the central beam, one must first describe what is meant by that term. The diameter of any circle bisects it. Two intersecting diameters establish the center of the circle at the point of their crossing. It is from this point that the central roentgen-ray beam is emitted. In the upper arch the central beam for all exposures should be directed at the apices of the teeth. As a general rule, an imaginary line drawn from the ala of the nose to the tragus of the ear will give the general apical line. In making all exposures the occlusal plane of the teeth must be perfectly horizontal and the sagittal plane of the face must be perpendicular to the occlusal plane of the teeth.

Upper Cuspid Region. In roentgenographing this region the following should be in evidence:

(1) The cuspid should be centered in the film.

(2) Mesially all or a good portion of the lateral incisor.

(3) Distally all or a good portion of the first bicuspid.

(4) The contact between the cuspid and lateral should be open (unless there is overlapping).

(5) The distal contact of the cuspid is usually found closed because of the superimposition of the shadow of the first bicuspid which is a wider tooth buccolingually.

(6) The nasal passage.

(7) The anterior wall of the maxillary sinus which joins to form the typical inverted "Y" that is so often in evidence in this region.

Two cotton rolls are again used as they were in the central lateral exposure. With the rolls placed between the film and the lingual surface of the crowns of the teeth and tissues, and the film parallel to the long axis of the cuspid, the tissue edge of the packet will be well back in the palate. The upper front or mesial corner will in many instances be closely adjacent to the opposite ridge. Care must be exercised to place the packet directly behind the cuspid tooth.

The vertical angulation is directed at right angles to the two parallel planes, and the horizontal angulation is directed so that the roentgen rays will pass through the height of contour on the labial surface of the cuspid crown before hitting the surface of the film. Usually this will bisect the angle formed by what is considered the corner of the dental arch.

In the posterior part of the upper arch a metal backing is used in conjunction with the film to insure its being kept parallel to the long axis of the teeth and to prevent any bending of the film. This backing consists of a piece of spring sheet metal, usually German silver, approximately the same size as the film. If the Bolin film is used, a shelf is bent at right angles to the plane of the metal in the horizontal direction upon which the film rests. This backing and the film are held in position by a film holder. A goiter hemostat, box lock 6½ inches long, makes an excellent holder. Over the beaks of this holder is placed a single-holed rubber cork which has previously been cut with a wet razor blade so that two of its sides are parallel, and which is used for the patient to bite upon. The backing is placed behind the film packet with the unbent edge slipped under the rubber jacket of the film. The opposite edge of the film rests against the shelf. With the film and backing in this position a full bite of the forceps is made as close to the shelf edge as possible. The handle of the film holder may then be rotated in the bite block so that the surface of the film may be placed parallel to the long axis of the teeth. The film is carried into the mouth in a horizontal position, and then rotated so that the tissue edge of the packet comes in contact with the palate in the proper position for the area being roentgenographed. At all times the bite block is kept against the incisal edge of the upper anterior teeth. The lower jaw bites firmly against the bite block, holding the film and film holder in the mouth and stabilizing the film placement.

Upper Bicuspid Region. The long axis of the first bicuspid is used as a guide in positionong the film with the correct inclination, unless this tooth is missing or malpositioned. In that case the inclination of the second bicuspid is used. The bite block is pushed up tightly against the edge of the film. When positioned in the mouth so that the plane of the film is parallel to the long axis of the teeth, the tissue edge of the packet will be against the palate, beyond and parallel to the mid-suture line, away from the teeth being roentgenographed. The packet should be kept forward in the mouth also, so that the distal contact of the cuspid is shown on the film. With the film in this position, the handle of the holder will protrude from the mouth in the region of the two central incisors. The handle of the film holder as it protrudes from the mouth is at right angles to the packet. Therefore, when the vertical angulation is directed parallel to the plane of the handle of the instrument, a good guide is obtained for correct vertical angulation and the need for looking into the mouth to check the inclination of the film is eliminated. In obtaining the horizontal angulation the roentgen rays are directed through the contact between the first and second bicuspids, or at right angles to the ridge in this area if one or both of the teeth are malpositioned, or if the area is edentulous. Upon this film should be shown the following:

(1) The distal contact of the cuspid.

(2) The first and second bicuspids, with their contacts open.

(3) The first molar and usually most of the second molar.

(4) A portion of the anterior wall and the floor of the maxillary sinus, depending on the latter's position and anterior extension in the body of the dental ridge.

Upper Molar Region. In roentgenographing the upper molar region three different exposures are necessary to adequately portray all of the anatomical landmarks. The first of these is known as the lateral or first molar projection and upon this film the following should be in evidence.

(1) The distal half of the second bicuspid.

(2) The first and second molars.

(3) The third molar if fully erupted and in position.

(4) The maxillary tuberosity and the floor of the maxillary sinus.

(5) The hamular process of the sphenoid bone, and a portion of the coronoid process of the mandible.

The contacts between the second bicuspid and the first molar, and the second and third molars may be open; usually, however, the contact between the first and second molars will be closed. The film and backing are held by the film holder in the same manner as for the bicuspid exposure, except the rubber bite block is pulled back ¼ inch from the edge of the film. When the handle of the holder is rotated in the bite block so that the surface of the film is properly inclined, the film is ready for placement in the mouth. The inclination of the first molar is used as a guide in film positioning. Generally, the alinement of the long axis of the first molar shows the crown tipped to the buccal. When positioned in the mouth so that the plane of the film is parallel with the long axis of the first molar, the tissue edge (the edge that comes in contact with the palate) is beyond and parallel to the mid-suture line, away from the teeth being roentgenographed. The packet should be placed far enough back in the mouth so that not more than the distal half of the second bicuspid is shown on the film. The vertical angulation is such that the roentgen rays are directed at right angles to the plane of the film. Again the handle of the film holder will be at right angles to the film packet; therefore, when the vertical angulation is directed parallel to the plane of the handle of the instrument a good guide is obtained for the correct vertical angulation. The horizontal angulation is directed at right angles to the dental ridge in the molar region. The central beam is directed at the apex of the first molar, just under the prominence of the zygoma.

Upper Second Molar Region. The upper second molar exposure is known as the mesial oblique projection and upon this film the following should be in evidence:

(1) The distal half of the first bicuspid.

(2) The second bicuspid.

(3) The first and second molars.

(4) Part of the third molar if it is erupted and in position.

(5) The floor of the maxillary sinus, above or closely adjacent to the apices of the roots.

The contacts between the first and second molars should be open; the other contacts may or may not be open. Because of the direction of the horizontal angulation, the maxillary tuberosity will usually not be shown. The film is retained in the film holder in exactly the same manner as described previously, the handle of the film holder being rotated in the bite block to obtain the proper inclination of the film. The inclination of the second molar is used as a guide in film positioning. Generally the alinement of the long axis of this tooth shows the crown tipped to the buccal. When positioned in the mouth properly, the tissue edge of the packet is beyond the median suture line. Instead of being parallel to the mid-suture line, the tissue edge of the film forms with it a triangle, the apex of which is in the back of the mouth. This is accomplished by placing the film well back in the mouth as if for a lateral molar projection, by using the back or distal corner as a pivot, shifting the front or mesial portion of the film laterally across the mouth. The front corner of the packet will be close to the opposite dental ridge in the region of the second bicuspid. The film holder as it protrudes from the mouth will be in the region of the cuspid tooth on the opposite side of the mouth. The plane of the film holder is at right angles to the plane of the film, and thus if the vertical angulation is parallel to that, it will be correct. The horizontal angulation is directed through the contact between the upper first and second molars. This contact is usually in a mesial oblique direction. The cen-

tral beam is again directed at the ala-tragus line just under the prominence of the zygoma.

Upper Third Molar Region. The third molar projection is known as the distal oblique exposure and is taken primarily to bring out the following structures:

(1) The distal half of the first molar.

(2) The second molar.

(3) All of the third molar regardless of its position in the tuberosity.

(4) The tuberosity.

(5) The distal wall and a portion of the floor of the maxillary sinus.

(6) The hamular process of the sphenoid bone.

(7) A portion of the coronoid notch.

(8) The coronoid process of the ramus of the mandible.

Because of the direction of the horizontal angulation, usually all of the contacts are closed. The film and backing are held in the same manner as previously described for the other posterior projections. The inclination of the third molar is used as a guide in film positioning. Generally the inclination of the long axis of this tooth shows the crown tipped well to the buccal. When positoned in the mouth so that the plane of the film is parallel to the long axis of the third molar, the tissue edge of the packet is beyond the median suture line, forming an angle to it. The triangle which is formed this time is just the opposite of that formed in the mesial oblique projection with the apex of the triangle in the *front* of the mouth. The procedure for obtaining this is similiar to that of the previous positioning, except that the front or mesial corner of the film is used as the pivot. The distal or back corner of the film will rest against the tuberosity on the opposite side of the mouth. When the film is in the correct position the handle of the instrument will protrude from the mouth in the region of the cuspid on the same side of the teeth being roentgenographed. By using the entire width of the distal portion of the upper arch for film placement, it is thus possible to project all of the maxillary tuberosity on the film without using extreme vertical angulation, and without obscuring the structures and tissues present with the shadow of the zygomatic arch and process. The vertical angulation is directed at right angles to the plane of the film. The horizontal angulation is directed downward and forward just under the inferior border of the zygomatic arch, in a distal oblique direction toward the film. This notch can be felt on the side of the face about the width of a finger anterior to the condylar joint. The central beam is directed at the ala-tragus line just under the prominence of the zygomatic arch in the region of the third molar.

Lower Central Lateral Incisor Region. In the lower arch most of the films are again placed in such a manner that the object-film distance is greater than accomplished in most other techniques. The lower central lateral region should portray the following anatomical structures:

(1) The central and lateral centered on the film with the contacts open (unless the teeth are overlapped).

(2) The mesial third of the cuspid.

(3) The mesial contact of the opposite central, if the mesio-distal width of the teeth will permit.

(4) Sometimes the genial tubercle, shown below the roots of the centrals (ordinarily the mental ridge and the inferior border of the mandible do not show unless the superior-inferior dimension of the jaw is very short).

To obtain images of these structures satisfactorily the narrow film is used in this region. Two cotton rolls are placed side by side on the tube side of the film, the ends flush with the bottom edge of the packet. The side of the index finger is placed in the mouth behind the film and under the tongue to compress the tissues in the floor of the mouth. This pressure on the tissues allows the packet to be carried deeper behind the teeth. With the index-finger rigid, almost its entire length is placed diagonally across the back of the film, avoiding a lateral pressure against the

film. The finger acts only as a prop, and the springiness of the cotton rolls keeps the film away from the teeth and parallel to their long axes. A third cotton roll to bite on is placed on the upper back or distal corner of the film packet to prevent lateral tipping, and to push the film even deeper into the mouth. The patient is then instructed to bite, which will stabilize the film retention.

The inclination of the central and lateral is the guide in film positioning unless there is some malpositioning. In that case, the tooth with the most typical alinement is used. With the two cotton rolls between the film and the crowns of the lingual surfaces of the teeth, the tissue edge of the packet will be well back in the mouth under the tongue. In most instances there should be at least an inch, or slightly more, between the top of the film and the incisal edge of the teeth. The film packet must be directly behind the teeth in the direction of the horizontal angulation, and not more than ¼ inch of film should be showing above the incisal edges of the teeth when sighting in the direction of the vertical angulation. In all lower exposures it will be necessary to change the head position from that used in the upper arch so that the occlusal plane of the teeth is horizontal. The sagittal plane of the face must always be at right angles to the occlusal plane. The vertical angulation is directed at right angles to the two parallel planes; i.e. the plane of the film and those of the long axes of the teeth. The horizontal angulation is directed so that the roentgen rays pass through the contact between the central and lateral, or at right angles to the ridge if there is excessive overlapping or if the area is edentulous. The central beam should be directed at the apices of the teeth, usually about ½ inch above the inferior border of the mandible in the male and ¼ inch above in the female.

Lower Cuspid Region. Placing the film for the cuspid region in the lower arch is accomplished in exactly the same manner as in the lower central-lateral region, except that the film is placed directly behind the cuspid. Upon this film the following should be shown:

(1) The cuspid centered on the film.

(2) Mesially all or a good portion of the lateral.

(3) Distally all or most of the bicuspid.

The contact between the cuspid and lateral should be open, unless there is overlapping, while the distal contact of the cuspid is usually found closed because of superimposition of the shadow of the first bicuspid, which is a wider tooth buccolingually. The roentgen rays in the vertical are directed at right angles to the two parallel planes and the horizontal angulation is directed so that it passes through the height of contour of the cuspid tooth. The central beam passes through the apices of the teeth.

Lower Bicuspid Region. Using the film with the long dimension placed *vertically*, the lower bicuspid region may be adequately portrayed with little discomfort to the patient. Upon this film the following should be shown:

(1) The distal contact of the cuspid.

(2) The first and second bicuspids, and all or most of the first molar.

(3) The mental foramen and the anterior portion of the mandibular canal, which terminates in the mental foramen.

Unless there is overlapping of the teeth, all of the contacts should be shown open since their direction is at right angles to the dental ridge. Because of the vertical angulation employed, the first molar is usually shown to be slightly foreshortened.

Three cotton rolls and the index finger are used to maintain the film in the proper position in the mouth. Two cotton rolls, side by side, are placed between the tube surface of the film and the teeth and the tissues. The third cotton roll is used to bite on and is placed obliquely across the upper back corner of the film to prevent lateral tipping, and to sink the packet deeper into the mouth. The side of the patient's index finger, held rigid, is placed diagonally across the back of the film so that it will compress the tissue in the floor of the

mouth and secure greater depth. The finger should not exert any lateral pressure against the back of the film, as its only function is that of a prop. The springy action of the two cotton rolls holds the film away from the teeth and parallel to their long axes. The patient bites gently on the cotton roll to stabilize the film retention. The inclination of the first bicuspid, or of the second bicuspid if it is the more typical in alinement in the dental arch, is used as a guide in film positioning. Generally the alinement of the first bicuspid shows the crown tipped slightly to the buccal. The film and rolls are placed in the mouth so that the tissue edge of the packet is under the tongue, well away from the teeth, and far enough forward to secure the distal contact of the cuspid.

In the usual case, the tissue edge or lower front corner of the film will be against the dental ridge of the opposite side. The forward edge of the film will be in contact with the opposite first bicuspid tooth. It is vital to emphasize that only by placing the film in this position can images of the apices of the teeth be obtained without using excessive minus vertical angulation. Through this placement, the film will be forward in the arch securing the distal contact of the cuspid without resorting to film bending. The vertical angulation should be directed so that the roentgen rays are at right angles to the plane of the film and the horizontal rays are directed through the contact between the first and second bicuspids. If the contact is closed due to excessive overlapping the rays should be directed at right angles to the ridge in this area. The central beam should again be directed at the apices of the teeth.

Lower Molar Region. It will be noted that in all previous areas of the mouth the film packet has been positioned *away* from the teeth and tissues. This was accomplished to make it possible for the operator to position the film parallel to the long axis of the teeth, thus obtaining anatomical accuracy. The lower molar region is the one and only exception to this rule. In this area the film can be placed against the dental ridge and still be parallel to the long axis of the teeth. To obtain the most satisfactory results it is necessary to make two exposures. The first of these being known as the lateral or first molar projection shows the following:

(1) The distal half of the second bicuspid.

(2) The first and second molars.

(3) A portion or all of the third molar.

(4) The mandibular canal in this area, and a portion of the retromolar triangle.

(5) The internal and external oblique ridges superimposed on one another.

Unless one or more of the teeth are malpositioned in the arch, the contacts should be shown open. The patient's index finger and one cotton roll are used to hold the film in place and keep it parallel to the long axis of the teeth. The side of finger is placed diagonally across the back of the film with the end of the finger deep in the reflex. The back of the packet may have a tendency to tip up on occasions, so a cotton roll is placed obliquely across the upper distal corner of the film. When the patient bites the cotton roll gently, the back portion of the film is then held down in place and the entire film is stabilized in position. The inclination of the long axis of the first molar is used as a guide in film positioning. Clinical evidence reveals that in the majority of cases, the inclination of this tooth is toward the lingual. The vertical angulation is directed so that the roentgen rays pass at right angles to the plane of the film. Since the inclination in most cases is toward the lingual, many times a *plus* vertical angulation will be necessary. The horizontal angulation should direct the rays through the contact between the first and second molars or at right angles to the ridge if the area is edentulous. The central beam should be directed at the apices, at a point midway between the distal root of the first molar and the mesial root of the second molar.

Lower Molar Region—Second Exposure. The second molar exposure in the lower arch is known as the distal oblique or third

molar projection and should portray the following structures:

(1) The distal half of the first molar.

(2) The second molar.

(3) The retromolar triangle and a portion of the ascending ramus.

(4) All of the third molar regardless of its position in the mandible.

(5) The mandibular canal and the internal and external oblique ridges superimposed one on another.

Because of the horizontal angulation, usually all of the contacts are closed. The film is retained in the same manner as that of the lateral molar projection. The inclination of the long axis of the third molar is used as a guide in film positioning. In many instances the long axis of the third molar is inclined in such a way that the crown tips toward the lingual, usually even more so than either the first or second molars. If the third molar is unerupted, imbedded, or impacted, then its relative position in the arch is usually higher, its crown being approximately on a level with the gingival third of the second molar roots. The film placement in this area is again the exception to the rule, the packet being placed well back in the reflex between the dental ridge and the tongue. If possible anatomically, the front edge of the film should be even with the lingual groove on the crown of the first molar. The vertical angulation should be directed in such a manner that the roentgen rays are at right angles to the surface of the film. The horizontal angulation should be such that the rays are directed toward the film in a distal oblique direction. The degree of angulation will depend upon how far back the film can be placed in the mouth, which is determined by the age of the patient, and the relative position of the third molar in the arch. The reason for projecting the rays with the vertical and horizontal angulation as indicated is to cast the shadows of the structures and tissues in this area downward and forward on the film.

Bite Wing Exposures. Most early carious lesions are more easily ascertained through the use of the bite wing film, rather than in apical exposures. The films are taken in such a manner that the central roentgen-ray beam passes directly through the contact points of the teeth, all effort being exerted to show the mesial and distal surfaces of the teeth in question. It is for this reason that it is important in a complete examination to include the bite wing film. However, since these surfaces are not parallel in the entire posterior region, it is necessary to make two exposures. In the bicuspid region the film should show:

(1) Mesially the distal halves of the cuspids.

(2) Distally, all or a good portion of the second molars.

(3) The crowns of the upper and lower teeth present.

(4) The crest of the dental ridges.

(5) The coronal third of the roots.

The film is placed in position well forward in the lower jaw, so that the distal portions of the cuspids will be shown. The tab rests on the occlusal surface of the lower teeth. The lower jaw is closed *slowly*, avoiding a protrusive or lateral bite, until the upper and lower teeth in centric hold the tab firmly. The operator's fingers are used to maintain control of the tab and film until the teeth are in occlusion. The upper and lower occlusal planes when in centric must be horizontal; the sagittal plane of the face must be perpendicular to this. The bite wing exposure is the only one in which a definite angulation is used, and here in the bicuspid region a plus 10 degree vertical angulation is employed in the average case. If the patient presents a cross-bite or edge-to-edge bite, a plus 12 degree vertical angulation is used. The horizontal angulation is directed so that the rays pass through the contact of the upper and lower first or second bicuspids, whichever teeth are more typically aligned in the arch. In this area the contacts usually run at right angles to the dental ridge. The central beam is directed at the occlusal surfaces in contact. The lip line, or corner of the mouth, is a good general guide.

In the molar region the film should be placed so that:

(1) Mesially nothing farther forward than the distal portions of the second bicuspids appear.

(2) Distally the entire surfaces of the third molars if they are in position.

The film is placed in the mouth in exactly the same manner as was the case for the bicuspid exposure, care being exercised to note that the patient is in tight centric relationship. Because of the usual lingual inclination of the lower molars, a plus 12 degree vertical angulation is used in normal cases and a plus 14 degree angulation if the patient presents a cross-bite or edge-to-edge bite. The horizontal angulation is directed so that the rays pass between the upper first and second molars, usually in a mesial oblique direction. The central beam is directed in the same manner as for the bicuspid projection.

CONCLUSION

There are many types of individuals in the practice of dentistry today. Some are chiefly conscious of the time element involved in each operation; others are chiefly conscious of the expense involved in various procedures; a few are really conscious of the service they may be able to render their patients. It will be noted that in this technique more exposures are required than in the commonly practiced techniques, and a greater length of time will also be necessary. However, if we are to render a diagnostic service to our patients which will provide us with images of conditions as they actually exist in the mouth, then a technique such as this for making exposures must be standardized and consistently used. Since intra-oral roentgenograms are a vital link in the chain of oral diagnosis the production of the most accurate images upon a dental film is of utmost importance. Most roentgenograms produced in dental offices today do not show these conditions, and consequently are of little value, leading only to faulty diagnosis. When a greater interest and effort is placed upon the type of intra-oral roentgenograms produced then more adequate roentgenograms will result.

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EDITORIALS

IS ROENTGEN THERAPY HAZARDOUS IN BENIGN BONE LESIONS?

DURING recent years several investigators have advanced the theory that roentgen therapy has a certain contributory role in the transformation of benign giant cell tumor of bone into osteogenic sarcoma and that it is therefore a hazardous procedure to use in benign bone lesions. To support this view much emphasis is laid on a series of cases collected from the early literature in which roentgen therapy was given for tuberculous arthritis and in which osteogenic sarcoma developed later within the treated area. Likewise, considerable importance is given to the observation made in conjunction with the radium dial painters in which, as is known, osteogenic sarcoma of one or several bones constituted a not infrequent terminal complication of the radium poisoning. Finally, individual cases are cited in which osteogenic sarcoma arose from bones which were in the field of roentgen therapy given for some independent lesion. In this latter group, severe radiation osteitis, due to over-irradiation, usually preceded for long periods the appearance of the sarcomatous transformation.

In discussing the attributed role of roentgen therapy in the sarcomatous conversion of a benign giant cell tumor, it is advisable to consider separately the occurrence of the possible malignant change without roentgen treatment and the effect of the roentgen rays on the bone as part of a routine irradiation.

The idea that giant cell tumor of bone is always benign originated in 1920 when the Registry of Bone Sarcoma coined for its designation the term of "benign giant cell tumor," the purpose being to eliminate the misleading name of "giant cell sarcoma" which was in vogue at that time. It was soon found, however, that a certain percentage of the giant cell tumors behaved as malignant from the beginning and that others became so later. This observation then has led to the assumption that there must be various causes which are responsible for the malignant transformation. Among these causes an ever increasing role was attributed to the disruptive action of the roentgen rays.

In the November issue of this JOURNAL[1] a study was published in which the various possibilities leading to a malignant degeneration of giant cell tumor of bone were analyzed on the basis of a personal series of the authors and an exhaustive review of the literature was given. The conclusions were reached that a malignant degeneration may be observed to occur: (1) spontaneously, if no treatment was given; (2) following repeated surgery and irradiation with different agents; (3) following roentgen therapy alone, and (4) following surgery alone when bone chips were used to fill the bone cavity. For obvious reasons, the group of cases of spontaneous malignant degeneration is too small to permit an evaluation of its exact incidence. In those cases in which various forms of treatment were used, the incidences were approximately the same. There was no evidence that in the series treated by roentgen therapy alone, or in association with other methods, the incidence of malignant degeneration was higher than if no roentgen therapy was employed. As a whole, it ap-

[1] Leucutia, T., and Cook, J. C., Malignant degeneration of benign giant cell tumor of bone. AM. J. ROENTGENOL. & RAD. THERAPY, November, 1949, *62*, 685–706.

peared that malignant degeneration of giant cell tumor of bone constitutes a phase in the natural sequence of events unaffected by the type of treatment given. The number of cases in which such malignant degeneration occurs is given as varying between 8 and 15 per cent.

The situation is somewhat similar to that observed in osteochondroma, where likewise a malignant degeneration is often found after long periods of quiescence and apparently without any demonstrable cause. In fact, it may be said that every benign tumor has its malignant counterpart, the time and frequency of the metaplasia depending on the individual characteristics of the tumor. A sarcomatous transformation is even noted in some of the more generalized diseases or anomalies of the osseous skeleton, as, for example, in hereditary chondrodysplasia, in fibrous dysplasia of bone, in myositis ossificans and osteitis fibrosa cystica. Above the age of fifty, Paget's disease is the most important contributory cause in a large percentage of the osteogenic sarcomas. It is highly improbable that the administration of roentgen therapy would have a tendency to further increase the incidence of malignant transformation in any of these conditions.

The effect of roentgen rays on bone depends, as in any other structure, on the amount given. A sharp distinction must be made between those cases which were grossly over-irradiated and those which received the customary therapeutic dose.

The observation that grossly over-irradiated tissue finally undergoes malignant neoplasia is as old as the science of radiology. If the action of the roentgen rays is confined mostly to the surface layers of the body, the changes occur primarily in the skin, eventually culminating in carcinoma. If the effect is deeper, changes may also be observed in the connective tissue elements resulting in sarcoma of the soft parts or bone. The ratio of carcinoma and sarcoma induced by roentgen rays is approximately ten to one. If the irradiation is internal, as, for example, in the case of the radium dial painters, and an irreversible deposition of the radioactive material occurs in the bone, osteosarcoma develops in a large majority of the cases. It is of fundamental importance, however, that in all these instances severe alterations of the tissues precede for years the malignant metaplasia. Marked atrophy of the skin, telangiectasis, ulceration, secondary infection, chronic osteitis, progressive bone absorption and other similar structural changes are nearly always present in one form or another. It is of no particular significance whether these alterations have been produced by a large single dose or by small doses repeated at shorter or longer intervals or by minimal doses acting continuously over a long period of time, as in the case of radium. Once the tissues have been severely damaged, no measure known at the present time can be used to preclude the eventual malignant transformation which follows in an impressive number of cases. The great majority of the radiation induced osteogenic sarcomas reported in the literature and all the osteosarcomas observed in the radium dial painters are the result of such over-irradiation.

In the ordinary roentgen treatment of giant cell tumor of bone and some of the allied conditions the required dose is considerably smaller. Permanent functional and anatomic results can be obtained with hardly any skin change being noticeable in later life. There is no evidence that under such conditions roentgen therapy constitutes a greater hazard in lesions of the bone than of any other structures. The increased ionization resulting from the somewhat greater absorption of the roentgen rays in the bone is an asset rather than a liability, permitting a slightly more efficient irradiation. This factor cannot be made responsible for any sarcomatous induction or conversion. It is more likely that osteogenic sarcomas which develop in giant cell tumors treated by roentgen therapy or arise after many years at the sites of giant cell tumors apparently cured by ordinary doses of roentgen rays represent samples of malig-

nant neoplasia occurring during the natural sequence of events. This view is supported by the fact that one hardly ever sees an osteogenic sarcoma develop in the massive bones of the pelvic girdle when radiation therapy is given for carcinoma of the cervix or some other intrapelvic lesion although the doses are considerably larger than those used in the benign tumors of bone.

It may be concluded from the above that roentgen therapy with ordinary doses is a safe procedure for the treatment of benign lesions of bone. The malignant transformation which is observed in a certain percentage of cases cannot be attributed to a direct cancerogenic effect of the roentgen rays, but is an occurrence in the natural sequence of events or represents a sequela of severe tissue damage produced by gross over-irradiation. This observation does not apply to internal irradiation with radioactive isotopes some of which possess, by their chemical nature, direct cancerogenic properties.

JOHN REMER
1862-1949

DR. JOHN REMER, of Charlotte, North Carolina, died on September 28, 1949, of a bronchogenic carcinoma in the right upper lobe.

John Remer was born October 21, 1862, in San Francisco, the son of Simeon Remer and Mary Farrell Remer. He received the degree of A.B. in 1886 and A.M. in 1889,

John Remer

both from Allegheny College. In 1890 he received his M.D. from the College of Physicians and Surgeons, Columbia University. He spent twenty-one years in general practice in New York City, and delivered some twelve hundred infants, before he specialized in radiology. His first published paper was in 1912, "The X-ray in General Dentistry." He soon found radiation therapy of more interest than diagnostic roentgenology, and he became associated with Dr. George M. MacKee. From dermatologic therapy he soon went on to the use of all types of radiation therapy. During World War I, he taught roentgen therapy in the school which the Army established in New York. He was co-author with Dr. W. D. Witherbee of a book "X-ray Dosage in Treatment," published in 1923 by Macmillan.

Dr. Remer was in charge of therapy or (later) consultant at New York Hospital for thirty-five years. He served on the staffs also of Vanderbilt Clinic, Harlem, Fordham, Governeur, St. Clare's, and Port Chester Hospitals. He was a fellow of the American Medical Association, of the New York Academy of Medicine, and of the American College of Radiology. He was a life member of the American Roentgen Ray Society, a member of the Radiological Society of North America, and of the New York Roentgen Society; and an honorary member of the Canadian Radiological Society.

In 1922 he was married to Miss Marie Donaldson, of New York, who survives him. He was a 32d degree Mason, and a life member of Harlem Lodge. He was a member of Theta Delta Chi and of the Columbia Club.

Dr. Remer practiced his profession until he was eighty-two. At the time of his retirement, some of his New York colleagues held a farewell party for him, in the old Murray Hill Hotel. He and Mrs. Remer first tried Sarasota, Florida, but found the summers too hot and the sunlight too bright for comfort. The more temperate climate of North Carolina was more to their liking. To a remarkable degree, John Remer fulfilled the old axiom "To win friends, show yourself friendly." When I last saw him, the day before his death, he was still the same winsome character who had won the love of all who knew him.

ROBERT H. LAFFERTY, M.D.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

MEETINGS OF ROENTGEN SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. Barton R. Young, 3401 North Broad St., Philadelphia, Pa. Annual meeting: Jefferson Hotel, St. Louis, Mo., Sept. 26–29, 1950.

American Radium Society
Secretary, Dr. H. F. Hare, 605 Commonwealth Ave., Boston, Mass. Annual meeting: Biltmore Hotel, New York, N. Y., May 25–26, 1950.

Radiological Society of North America
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y. Annual meeting: Cleveland Auditorium and Statler Hotel, Cleveland, Ohio, Dec. 4–9, 1949.

American College of Radiology
Executive Secretary, William C. Stronach, 20 N. Wacker Drive, Chicago 6. Annual meeting: 1950, to be announced.

Section on Radiology, American Medical Association
Secretary, Dr. Paul C. Hodges, 950 E. 59th St., Chicago, Ill. Annual Meeting: 1950, to be announced.

Alabama Radiological Society
Secretary, Dr. W. W. Anderson, Tuscaloosa, Ala. Meets time and place Alabama State Medical Association.

Arizona Association of Pathologists and Radiologists
Secretary, Dr. R. Lee Foster, 507 Professional Bldg., Phoenix, Ariz. Two regular meetings a year. The annual meeting at time and place of State Medical Association and interim meeting six months later.

Arkansas Radiological Society
Secretary, Dr. Fred Hames, 511 National Bldg., Pine Bluff, Ark. Meets every three months and also at time and place of State Medical Association.

Atlanta Radiological Society
Secretary, D. W. W. Bryan, 490 Peachtree St., N.E., Atlanta, Ga. Meets monthly, except during three summer months, on second Friday evening.

Brooklyn Roentgen Ray Society
Secretary, Dr. J. J. Daversa, 345 75th St., Brooklyn, N. Y. Meets monthly fourth Tuesday, Oct. through April.

Buffalo Radiological Society
Secretary, Dr. Mario C. Gian, 610 Niagara St., Buffalo, N. Y. Meets second Monday evening each month, October to May inclusive.

Central New York Roentgen Ray Society
Secretary, Dr. Dwight V. Needham, 608 E. Genesee St., Syracuse N. Y. Meets January, May, November.

Central Ohio Radiological Society
Secretary, Dr. Paul D. Meyer, Grant Hospital, Columbus, Ohio. Meets at 6:30 P.M. on second Thursday of October, December, February, April, and June at Seneca Hotel, Columbus, Ohio.

Chicago Roentgen Society
Secretary, Dr. John H. Gilmore, 720 N. Michigan Ave., Chicago 11, Ill. Meets second Thursday of each month October to April inclusive at the University Club.

Cincinnati Radiological Society
Secretary, Dr. E. C. Elsey, 927 Carew Tower, Cincinnati 2, Ohio. Meets last Monday of each month, September to May, inclusive.

Cleveland Radiological Society
Secretary, Dr. J. R. Hannan, Cleveland Clinic, Cleveland 6, Ohio. Meetings at 6:30 P.M. on fourth Monday of each month from October to April.

Colorado Radiological Society
Secretary, Dr. Paul E. RePass, 306 Republic Bldg., Denver 2, Colo. Meets third Friday of each month at University of Colorado Medical Center or at Denver Athletic Club.

Connecticut Valley Radiologic Society
Secretary, Dr. E. W. Godfrey, 1676 Boulevard, West Hartford, Conn. Meets second Friday Oct. and April.

Dallas-Fort Worth Roentgen Study Club
Secretary, Dr. X. R. Hyde, Medical Arts Bldg., Fort Worth, Texas. Meets in Dallas on odd months and in Fort Worth on even months, on third Monday, 7:30 P.M.

Detroit Roentgen Ray and Radium Society
Secretary, Dr. W. G. Belanger, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne County Medical Society Building.

East Bay Roentgen Society
Secretary, Dr. Dan Tucker, 434-30th St., Oakland 9, Calif. Meets first Thursday each month at Peralta Hospital, Oakland.

Florida Radiological Society
Secretary, Dr. F. K. Hurt, Riverside Hospital, Jacksonville, Fla. Meets twice annually, in the spring with the annual State Society meeting, and in the fall.

Georgia Radiological Society
Secretary, Dr. Robert Drane, DeRenne Apartments, Savannah, Ga. Meets in mid-winter and at annual meeting of Medical Association of Georgia in the spring.

Greater Miami Radiological Society
Secretary, Dr. David Kirsh, 712 duPont Bldg., Miami 32, Florida. Meets last Wednesday of each month at 8:00 P.M. at Veterans Administration Regional Office.

Houston X-ray Club
Secretary, Dr. Curtis H. Burge, 3020 San Jacinto St. Houston 4, Texas. Meets fourth Monday each month.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Illinois Radiological Society
Secretary, Dr. Wm. DeHollander, St. John's Hospital, Springfield, Ill. Meets three times a year.

Indiana Roentgen Society
Secretary, Dr. William M. Loehr, 712 Hume-Mansur Bldg., Indianapolis 4. Meets second Sunday in May.

Iowa X-Ray Club
Secretary, Dr. Arthur W. Erskine, 326 Higley Bldg., Cedar Rapids, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. Special meetings by announcement.

Kansas Radiological Society
Secretary, Dr. Anthony F. Rossitto, Wichita Hospital, Wichita, Kan. Meets annually with State Medical Society.

Kentucky Radiological Society
Secretary, Dr. W. C. Martin, 321 W. Broadway, Louisville. Meets annually in Louisville on first Saturday in Apr.

Kings County Radiological Society
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Los Angeles Radiological Society
Secretary, Dr. Wybren Hiemstra, 1414 S. Hope St., Los Angeles 15, Calif. Meets second Wednesday each month at Los Angeles County Medical Assn. Building.

Louisiana Radiological Society
Secretary, Dr. J. R. Anderson, 1130 Louisiana Ave., Shreveport. Meets annually during Louisiana State Medical Society Meeting.

* Secretaries of societies are requested to send timely information promptly to the Editor.

LOUISVILLE RADIOLOGICAL SOCIETY
Secretary, Dr. E. L. Pirkey, Louisville General Hospital, Louisville 2, Ky. Meets monthly on second Friday at Louisville General Hospital.

MICHIGAN ASSOCIATION OF ROENTGENOLOGISTS
Secretary, Dr. R. D. McDuff, 220 Genesee Bank Bldg., Flint 3, Mich.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary, Dr. T. J. Pfeffer, 839 N. Marshall St., Milwaukee 2, Wis. Meets monthly on second Monday at University Club.

MINNESOTA RADIOLOGICAL SOCIETY
Secretary, Dr. Chauncey N. Borman, 802 Medical Arts Bldg., Minneapolis 2, Minn. Two meetings yearly, one at time of Minn. State Med. Assn., the other in the fall.

NEBRASKA RADIOLOGICAL SOCIETY
Secretary, Dr. Ralph C. Moore, Nebraska Methodist Hospital, Omaha 3, Nebr. Meets third Wednesday of each month, at 6 P.M. at either Omaha or Lincoln.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. George Levene, Massachusetts Memorial Hospitals, Boston, Mass. Meets monthly on third Friday, Harvard Club.

NEW HAMPSHIRE ROENTGEN RAY SOCIETY
Secretary, Dr. A. C. Johnston, Elliott Community Hospital, Keene, N. H. Meets four to six times yearly.

NEW YORK ROENTGEN SOCIETY
Secretary, Dr. F. H. Ghiselin, 111 East 76th St., New York 21, N. Y. Meets monthly on third Monday, New York Academy of Medicine, at 8:30 P.M.

NORTH CAROLINA RADIOLOGICAL SOCIETY
Secretary, Dr. J. F. Hemphill, 1420 E. Fifth St., Charlotte 4, N. C. Meets in May and October.

NORTH DAKOTA RADIOLOGICAL SOCIETY
Secretary, Dr. C. O. Heilman, 807 Broadway, Fargo. Meetings held by announcement.

NORTHERN CALIFORNIA RADIOLOGICAL CLUB
Secretary, Dr. R. L. Ayers, 726 4th St., Marysville, Calif. Meets at dinner last Monday, every second month, except June, July and August.

OHIO STATE RADIOLOGICAL SOCIETY
Secretary, Dr. E. C. Elsey, 927 Carew Tower, Cincinnati 2, Ohio.

OKLAHOMA STATE RADIOLOGICAL SOCIETY
Secretary, Dr. W. E. Brown, Tulsa, Okla. Three regular meetings annually.

OREGON RADIOLOGICAL SOCIETY
Secretary, Dr. Selma Hyman, Univ. of Oregon Medical School, Portland, Oregon. Meets monthly 2nd Wednesday, 8:00 P.M.

ORLEANS PARISH RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets first Tuesday of each month.

PACIFIC NORTHWEST RADIOLOGICAL SOCIETY
Secretary, Dr. S. J. Hawley, 1320 Madison St., Seattle 4, Wash. Meets annually in May.

PACIFIC ROENTGEN SOCIETY
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

PENNSYLVANIA RADIOLOGICAL SOCIETY
Secretary, Dr. J. M. Converse, 416 Pine St., Williamsport. Annual Meeting: May 20 and 21, 1949, Bedford Springs Hotel, Bedford, Pa.

PHILADELPHIA ROENTGEN RAY SOCIETY
Secretary, Dr. G. P. Keefer, 1930 Chestnut St. Meets first Thursday each month October to May, at 8:00 P.M., in Thomson Hall, College of Physicians.

PITTSBURGH ROENTGEN SOCIETY
Secretary, Dr. E. J. Euphrat, 3500 Fifth Ave., Pittsburgh 13, Pa. Meets 6:30 P.M. at Webster Hall Hotel on second Wednesday each month, October to May inclusive.

QUEENS ROENTGEN RAY SOCIETY
Secretary, Dr. J. E. Goldstein, 88-29 163rd St., Jamaica 3, N. Y. Meets fourth Monday of each month except during the summer.

RADIOLOGICAL SECTION, BALTIMORE MEDICAL SOCIETY
Secretary, Dr. Harry A. Miller, 2452 Eutaw Place, Baltimore. Meets third Tuesday each month, Sept. to May.

RADIOLOGICAL SECTION, CONNECTICUT MEDICAL SOCIETY
Secretary, Dr. Fred Zaff, 135 Whitney Ave., New Haven, Conn. Meets bimonthly on second Wednesday.

RADIOLOGICAL SECTION, DISTRICT OF COLUMBIA MEDICAL SOCIETY
Secretary, Dr. K. C. Corley, 1835 Eye St., N. W., Washington, D. C. Meets Medical Society Auditorium, third Thursday, Jan., March, May, Oct. at 8:00 P.M.

RADIOLOGICAL SECTION, SOUTHERN MEDICAL ASSOCIATION
Secretary, Dr. Roy G. Giles, Temple, Texas.

RADIOLOGICAL SOCIETY OF NEW JERSEY
Secretary, Dr. Benjamin Copleman, 280 Hobart St., Perth Amboy, N. J. Meets annually at Atlantic City at time of State Medical Society and mid-winter at Elizabeth, N. J.

ROCHESTER ROENTGEN RAY SOCIETY, ROCHESTER, N. Y.
Secretary, Dr. Ralph E. Alexander, 101 Medical Arts Bldg. Meets monthly on third Monday from October to May, inclusive, 8 P.M. at Strong Memorial Hospital.

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY
Secretary, Dr. Maurice D. Frazer, 1037 Stuart Bldg., Lincoln, Nebr. Meets Shirley-Savoy Hotel, Denver, Colo. August 18, 19, 20, 1949.

ST. LOUIS SOCIETY OF RADIOLOGISTS
Secretary, Dr. C. J. Nolan, 737 University Club Bldg., St. Louis 3, Mo. Meets fourth Wednesday each month, except June, July, August, and September.

SAN DIEGO ROENTGEN SOCIETY
Secretary, Dr. R. F. Niehaus, 1831 Fourth Ave., San Diego, Calif. Meets monthly, first Wednesday at dinner.

SECTION ON RADIOLOGY, CALIFORNIA MEDICAL ASSOCIATION
Secretary, Dr. D. R. MacColl, 2007 Wilshire Blvd., Los Angeles 5, Calif.

SECTION ON RADIOLOGY, ILLINOIS STATE MEDICAL SOCIETY
Secretary, Dr. Harold L. Shinall, St. Joseph's Hospital, Bloomington, Ill.

SHREVEPORT RADIOLOGICAL CLUB
Secretary, Dr. R. W. Cooper, Charity Hospital, Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M., September to May inclusive.

SOUTH CAROLINA X-RAY SOCIETY
Secretary, Dr. T. A. Pitts, Baptist Hospital, Columbia, S. C. Meets in Charleston first Thursday in November, also with South Carolina State Medical Association.

TENNESSEE RADIOLOGICAL SOCIETY
Secretary, Dr. J. M. Frère, 707 Walnut St., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

TEXAS RADIOLOGICAL SOCIETY
Secretary, Dr. R. P. O'Bannon, 650 Fifth Ave., Fort Worth 4, Texas. Next meeting, Dallas, Texas, February 3 and 4, 1950.

UNIVERSITY OF MICHIGAN DEPARTMENT OF ROENTGENOLOGY STAFF MEETING
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

UNIVERSITY OF WISCONSIN RADIOLOGICAL CONFERENCE
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets first and third Thursdays 4:00 to 5:00 P.M., September to May inclusive. Room 203, Service Memorial Institute, 426 N. Charter St., Madison.

Utah State Radiological Society
Secretary, Dr. Angus K. Wilson, 343 S. Main St., Salt Lake City 1, Utah. Meets third Wednesday in September, November, January, March and May.

Virginia Radiological Society
Secretary, Dr. P. B. Parsons, Norfolk General Hospital, Norfolk, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. Homer V. Hartzell, 310 Stimson Bldg., Seattle 1, Wash. Meets fourth Monday each month, October through May, College Club, Seattle.

X-Ray Study Club of San Francisco
Secretary, Dr. W. F. Reynolds, University of California Hospital, San Francisco. Meets from January to July, 1949, at Lane Hall, Stanford University Hospital, and from July to December 1949, at San Francisco Hospital.

Cuba

Sociedad de Radiología y Fisioterapia de Cuba
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

Mexico

Sociedad Mexicana de Radiologia y Fisioterapia
General Secretary, Dr. D. P. Cossio, Marsella No. 11, Mexico, D. F. Meets first Monday of each month.

British Empire

British Institute of Radiology Incorporated with the Röntgen Society
Ordinary meeting, on the Thursday preceding the third Friday, October to May at 8:15 p.m.
Medical Members' meeting, on third Friday in each month at 5:00 p.m., 32 Welbeck St., London, W 1.

Faculty of Radiologists
Honorary Secretary, Dr. J. F. Bromley, 45, Lincoln's Inn Fields, London, W.C.2, England.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 p.m. at the Royal Society of Medicine, 1 Wimpole St., London.

Canadian Association of Radiologists
Honorary Secretary, Dr. E. M. Crawford, 1535 Sherbrooke St., West, Montreal 26, Que. Meetings January and June.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Française d'Electrologie et de Radiologie Médicales
Secretary, Dr. Origéne Dufresne, 4120 Ontario St., East, Montreal, P. Q.

Australian and New Zealand Association of Radiologists
Honorary Secretary, Dr. Alan R. Colwell, 135 Macquarie St., Sydney, N.S.W.
Honorary Secretaries, State Branches:
New South Wales, Dr. E. W. Frecker, 135 Macquarie St., Sydney.
Victoria, Dr. T. L. Tyrer, 3 Lockerbie Court, East St. Kilda.
Queensland, Dr. J. Adam, 131 Wickham Terrace, Brisbane.
South Australia, Dr. R. de G. Burnard, 170 North Terrace, Adelaide.
Western Australia, Dr. A. M. Nelson, 179-B St. Georges Terrace, Perth.
New Zealand, Dr. E. G. Lynch, 12 Bolton St., Wellington.

South America

Sociedad Argentina de Radiologia
Secretary, Dr. Guido Gotta, Buenos Aires, Argentina. Meetings are held monthly.

Sociedad Argentina de Radiología, Filial del Litoral
President, Dr. Francisco P. Cifarelli. Meets second Wednesday each month, at 7:00 p.m., at 663 Italia St. in Rosario.

Sociedade Brasileira de Radiologia Medica
Secretary, Dr. Nicola Caminha, Av. Mem de Sa, Rio de Janeiro, Brazil. Meets monthly, except during January, February and March.

Sociedade Brasileira de Radioterapia
Secretary, Dr. Oscar Rocha von Pfuhl, Av. Brigadeiro Luiz Antonio, 644 São Paulo, Brazil. Meets monthly on second Wednesday at 9 p.m. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Peruana de Radiologia
Secretary, Dr. Julio Bedoya Paredes, Apartado, 2306, Lima, Peru. Meets monthly except during January, February and March, at Asociación Médica Peruana "Daniel A. Carrión," Villalta, 218, Lima.

Sociedad de Radiologica, Cancerologia y Fisica Medica del Uruguay
Secretary, Dr. Arias Bellini.

Continental Europe

Société Belge de Radiologie
General Secretary, Dr. S. Masy, 111 Avenue des Alliés, Louvain, Belgium. Meets monthly, second Sunday at Maison des Médecins, Brussels.

Ceskoslovenská společnost pro röntgenologii a radiologii v Praze
Secretary, Dr. Roman Bláha, Praha XII, stát. nemocnice, Czechoslovakia. Meets monthly except during July, August, and September. Annual general meeting.

Polish Society of Radiology
Secretary, Dr. L. Zgliczynski, 59 Nowogrodzka St., Warsaw, Poland. Next meeting, Krakow, June 2 and 3, 1949.

Gdansk Section, Polish Society of Radiology
Secretary, Dr. A. Smigielska, Akademia Lekarska, Gdansk. Meets monthly last Sunday at 10.30, X-Ray Dept., Akademia Gdansk.

Warsaw Section, Polish Society of Radiology
Secretary, Dr. L. Zgliczynski, 59 Nowogrodzka St., Warsaw, Poland. Meets monthly.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banual Mărăcine, 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad.
USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

Leningrad Roentgen Ray Society
Secretaries, Drs. S. G. Simonson and G. A. Gusterin. Meets monthly, first Monday at 8 o'clock, State Institute of Roentgenology and Radiology, Leningrad.

Moscow Roentgen Ray Society
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky. Meets monthly, first Monday, 8 p.m.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

Sociedad Espanola de Radiologia y Electrologia
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7. Madrid, Spain. Meets monthly in Madrid.

Schweizerische Röntgen-Gesellschaft (Société Suisse de Radiologie)
President, Dr. H. E. Walther, Gloriastr. 14, Zürich, Switzerland.

Societa Italiana di Radiologia Medica
Secretary, Prof. Mario Ponzio, Ospedale Mauriziano. Torino, Italy. Meets biannually.

DEPTH DOSE DATA FOR RADIATION FROM 22 MEV. BETATRON

To the Editor:

H. E. Johns *et al.*[1] have recently published measurements of depth dose in a water phantom using 22 mev. betatron radiation filtered by a conical copper plug shaped to compensate for the normal angular distribution of the roentgen radiation and produce a uniform intensity over a selected field. We have read this article with a great deal of interest and it appears to be the result of considerable careful experimental work.

There is one statement in their article on page 262 in reference to the work of Charlton and Breed[2] with which we are not in agreement because it indicates a misinterpretation of the technical data. They state that the actual peak percentage depth dose they obtain at 22 mev. occurs at a considerably greater depth than that obtained by Charlton and Breed at 20 mev. Their statement should be further clarified to point out the essential difference between the two sets of measurements to which they refer. In the particular data in our article to which they refer, the maximum depth dose at 20 mev. occurred at 1.5 cm. in a pressboard phantom with unfiltered radiation. These particular depth dose data were taken with unfiltered radiation in order to cover with a single technique the voltage range from 20 to 100 mev. over most of which filtration gives only a slight advantage. On pp. 172–174 of the Charlton and Breed article there appears a discussion of the use of filters and curves shown that were taken with filtered radiation. This is the 20 mev. depth dose data that should be compared with the Johns *et al.* 22 mev. measurements. A comparison of these data makes it apparent that at 20 mev. there is excellent agreement between the two sets of data, particularly when it is remembered that our curves were obtained using a flat copper filter 1 cm. thick while the curves of Johns *et al.* were obtained with a compensating copper filter of apparently about 3 cm. maximum thickness. It is also interesting that the depth dose agreement is so close when one considers the fact that the results were obtained with two entirely different machines and by very different measuring techniques.

E. E. CHARLTON

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[1] Johns, H. E., Darby, E. K., Haslam, R. N. H., Katz, L., Harrington, E. L. Depth dose data and isodose distributions for radiation from a 22 mev. betatron. AM. J. ROENTGENOL. & RAD. THERAPY, 1949, *62*, 257–268.

[2] Charlton, E. E., and Breed, H. E. Some depth dose studies of roentgen rays for energy levels from 20 to 100 million electron volts. AM. J. ROENTGENOL. & RAD. THERAPY, 1948, *60*, 158–174.

RADIOISOTOPE TECHNIQUES COURSES

Some vacancies still remain for the winter series of three Radioisotope Techniques Courses offered by the Special Training Division of the Oak Ridge Institute of Nuclear Studies.

Interested persons who can attend a course during the winter months are urged to make application for one of these courses in view of the heavy load of applications from university people for participation in the summer courses. The courses will begin on January 2, January 30, and March 6, 1950. Applications should be made as soon as possible. Requests for application and additional information should be addressed to Dr. Ralph T. Overman, Chairman, Special Training Division, Oak Ridge Institute of Nuclear Studies, P. O. Box 117, Oak Ridge, Tennessee.

The courses are offered at Oak Ridge to acquaint research workers with the safe and efficient use of radioisotopes as tracers. Emphasis is placed on individual laboratory work, with sufficient lecture material included to provide the necessary background information. Eleven of the courses already have been given. Thirty-two participants can be accommodated in each of the three four-week courses.

GREATER MIAMI RADIOLOGICAL SOCIETY

The radiologists of the Greater Miami area, after meeting at monthly intervals informally for a period of about two years,

organized at a meeting held on October 26, 1949. The name, Greater Miami Radiological Society, was adopted and the following officers were elected: *President*, Dr. Gerard Raap; *Secretary*, Dr. David Kirsh. Meetings are held on the last Wednesday of the month throughout the year at 8:00 P.M. at the Veterans Administration Regional Office, 3300 N.E. Second Avenue, Miami, Florida.

CINCINNATI RADIOLOGICAL SOCIETY

The first annual meeting of the Cincinnati Radiological Society was held on Monday, October 17, 1949, and the following officers were elected for the coming year: *President*, Dr. Maurice R. Walsh; *Secretary*, Dr. E. C. Elsey.

NORTHERN CALIFORNIA RADIOLOGICAL CLUB

The officers of the Northern California Radiological Club for the year 1949–1950 are as follows: *President*, Dr. George Hemminger, Sacramento, California; *Secretary*, Dr. Robert L. Ayers, Marysville, California.

OREGON RADIOLOGICAL SOCIETY

At a recent meeting of the Oregon Radiological Society the following officers were elected for the coming year: *President*, Dr. Sherman E. Reese; *Vice-President*, Dr. G. B. Isenhart; *Secretary-Treasurer*, Dr. Selma Hyman.

PITTSBURGH ROENTGEN SOCIETY

At the meeting of the Pittsburgh Roentgen Society held on October 12, 1949, the following officers were elected for the coming year: *President*, Dr. R. Paul Meader; *Vice-President*, Dr. Eva S. Carey; *Secretary-Treasurer*, Dr. Edwin J. Euphrat.

ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

Department Editor: George M. Wyatt, M.D., 1835 Eye St., N.W., Washington 6, D. C.

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ROENTGEN DIAGNOSIS

HEAD

Buckley, Robert W., and Yakovlev, Paul I. Dysostosis of skull, face and extremities (acrocephalosyndactyly). *Am. J. Dis. Child.*, May, 1948, *75*, 688–695.

In connection with the study of a recently observed case of acrocephaly, facial deformities and syndactylism, with mental deficiency, the authors reviewed the literature pertaining to clinical descriptions of similar cases. The survey of the literature revealed numerous cases in which different features of their case were present in several combinations. Their patient's set of physical and mental anomalies would appear to represent a complete picture, parts of which have been reported under various clinical names and personal eponyms.

The roentgenograms of the patient, a boy of twelve, showed in the skull, a high crested appearance of the calvarium, the characteristic "lacunar" defects in the frontal region, shallow orbits, dental anomalies and prognathism. The spinous processes of the fifth and sixth cervical vertebrae were apparently fused. The head of the right humerus was translucent with a thin cortex and expanded and deformed outline. It appeared to be fused to the glenoid process of the scapula, with radiating dense bony spicules continuous with similar structures on the blade of the scapula. The distal extremity of the humerus showed normal density and persistent epiphyseal separation, the shaft being short and bowed. Posteroanterior views of both feet showed condensation of the phalanges of each digit, with preservation of the proximal epiphyses, but only indistinct separation distally. The first and second metatarsals were grossly deformed bilaterally. The left second metatarsal was angulated laterally with local density suggestive of old fracture, and the right first and second metatarsal were united by a broad, bony band with anomalous articulation of the first on the ankle. Roentgenograms of the left shoulder and hands showed analogous deformities.

The authors express the opinion that "Apert's disease," or acrocephalosyndactylism, and "Crouzon's disease," or craniofacial dysostosis, are both manifestations of the same pathologic process. The condition of faulty bone development and articulation is believed to be due to a germ-plasm defect influencing the growth of both "membranous" and "cartilaginous" bone. —*R. S. Bromer, M.D.*

Keleman, George, and Holmes, Edgar M. Cavernous haemangioma of the frontal bone. *J. Laryng. & Otol.*, Sept., 1948, *62*, 557–563.

The authors state that only 2 cases of cavernous hemangioma limited to the frontal bone have been reported and theirs is the third. They describe the characteristic "sunburst" pattern with trabeculations which radiate from a common center and mostly in the plane of the bone. Sometimes there are additional perpendicular striations or radiating spicules which are identical with those of an osteogenic sarcoma. In these cases an accurate diagnosis can be made only by histopathological examination.

They report a case in which repeated trauma is a factor and may have contributed to the progress of the lesion. They stress complete removal of both tables of the skull and a small surrounding area of normal bone to assure a complete cure. Recurrences after incomplete removal are common.—*F. M. Windrow, M.D.*

Mitchell, J. F. O., and Astley, Roy. Radiology of the temporal bone. *J. Laryng. & Otol.*, Sept., 1948, *62*, 564–567.

The authors present 14 good roentgenograms of a disarticulated temporal bone in the various usual projections. Wire markers outline the external and internal auditory meati, the eustacian tube and the facial nerve. Thin lead foil

is used to outline the sigmoid sinus groove and the mastoid antrum. The reproductions of the roentgenograms offer a good review of the roentgen anatomy of the temporal bone.—*F. M. Windrow, M.D.*

WELIN, SOELVE. The roentgen ray examination of the paranasal sinuses with particular reference to the frontal sinuses. *Brit. J. Radiol.*, Sept., 1948, *21*, 431–437.

The routine examination of the paranasal sinuses as carried out by the author consists of six projections. The direct posteroanterior view is made with the film angled 30 degrees from the vertical and with the patient's forehead against the film. A second posteroanterior view is made with the film tilted slightly less and with the nose and open mouth against the film. A direct lateral view is made. Two submentovertical views are made, one exaggerated so that the mandible is projected anterior to the frontal sinuses. This view permits visualization of the depth of the frontal cells, both medially and laterally, and shows well the thickness of the walls. These five views are all made with the patient upright. In this way fluid levels are usually shown, but at times the extent and location of fluid are better shown in the sixth view made in a posteroanterior projection with the patient horizontal and with either right or left side down. This also shows small amounts of fluid which might be invisible by other methods. Air trapped in hyperplastic or edematous mucosal folds has been demonstrated in this way.—*E. F. Lang, M.D.*

VERMUND, HALVOR. The relation of hypophysis to carbohydrate and basal metabolism. *Acta med. Scandinav.*, April, 1948, *131*, 515–546.

Report is made of a case in which the patient, a female, aged forty-six, had signs of acromegaly. Roentgenograms revealed a tumor of the pituitary. Three months before her hospital admission she developed symptoms of diabetes mellitus, and on examination in the hospital she had considerable acidosis with imminent coma. She recovered under therapy. Then on the twelfth day after admission, signs of hypoglycemia suddenly appeared. Insulin and dietary restrictions were withdrawn, and after this time there was no hyperglycemia and no glucosuria. The glucose tolerance test became normal, though previously diabetic in nature, and the diabetes was considered cured. Hypothyroid symptoms developed, necessitating use of thyroid extract. The signs of acromegaly regressed, and the patient has since been in good health and quite fit for work.

It is postulated that increased pituitary function led to acromegaly, and probably to the adverse carbohydrate change. Hyperfunction was followed by hypofunction, with regression of the acromegaly, depression of thyroid function and disappearance of the diabetogenic and insulin-antagonistic effects.—*Charles Nice, M.D.*

PENNYBACKER, JOE, and RUSSELL, DOROTHY S. Necrosis of the brain due to radiation therapy; clinical and pathological observations. *J. Neurol., Neurosurg. & Psychiat.*, Aug., 1948, *11*, 183–198.

The authors report the clinical and pathological features of 5 cases of necrosis of the brain following radiation therapy, these cases having been treated in different centers with accepted techniques and dosages.

The smallest dose utilized was 2,300 r in a single exposure to a basal cell carcinoma of the scalp (230 kv., 10 ma., half-value layer 1.3 mm. Cu, focal skin distance 29 cm., 7 cm. in diameter) and 4,800 mg-hr. of radium implantation (begun after neurological signs had already appeared). Large dosage was used in a spongioblastoma multiforme which received 44,400 mg-hr. at 4 cm. distance in 1935, through nine fields and then remained asymptomatic until 1941. In March, 1942, she received 9,900 r through five fields. She died in July, 1942, with no residual tumor at postmortem, but with extensive radiation degeneration.

The clinical effects may come on suddenly, progress to a point and become arrested; or they may come on gradually and lead to death. In each case, there was a long latent period between the radiation treatment and the onset of clinical signs of necrosis. The shortest interval was nine months, the longest five years.

The pathology of the necrosis appears to be related to reactions in the smaller blood vessels (perforating) in which collagenous thickening, fibrinoid necrosis and thrombosis are conspicuous.

As it seems impossible to predict in which cases necrosis will occur, it is suggested that irradiation should be reserved for inoperable tumors and those cases in which no further surgical procedures are contemplated.

The authors stress that these changes are not a contraindication to radiation therapy especially since the combination of surgery and irradiation resulted in the remarkable survival of over seven years in the spongioblastoma multiforme case quoted above.—*Herbert Lobsenz, M.D.*

Savitsky, Elias, and Spinelli, Vincent A. Agenesis of corpus callosum in infancy; clinical and roentgenologic aspects. *Am. J. Dis. Child.*, July, 1948, *76*, 109–115.

The case reported in this paper is the eleventh in which postmortem diagnosis and necropsy studies have been correlated. Their patient and that reported by Reeves are the youngest patients for whom the diagnosis of agenesis of the corpus callosum was made by air studies. Associated anomalies in this case were fetal arrangement of the medial sulci, microgyria, polygyria, heterotopia, internal hydrocephalus and granular ependyma.

The clinical picture of agenesis of the corpus callosum is nonspecific, since it probably depends on the pathologic disturbances that coexist with the condition, which vary from case to case.

The authors consider the combination of roentgenologic signs described by Davidoff and Dyke as diagnostic. These are:

1. Lateral ventricles are widely separated.
2. Dorsal margins of lateral ventricles are pointed or angular rather than flat.
3. Medial borders of lateral ventricles are concave.
4. Caudal portions of later ventricles are dilated.
5. Interventricular foramens are elongated.
6. The third ventricle is dilated and extends dorsally beyond the normal limits.
7. Air shadows cast by sulci on medial aspects of cerebral hemispheres show radial arrangement of sulci and their extension through the zone normally occupied by the corpus callosum.

The authors also quote Penfield and Hyndman who considered the bicornuate appearance of the bodies of the lateral ventricles as pathognomonic of the condition.—*R. S. Bromer, M.D.*

Mathis, William H., Jr., and Schulz, Milford D. Roentgen diagnosis of glomus tumors. *Radiology*, July, 1948, *51*, 71–76.

Tumors of the subcutaneous neuromyoarterial plexus, the glomus body, occur once in 32,000 hospital admissions, and once in 4,500 surgical specimens at the Massachusetts General Hospital. The glomus is a minute specialized body which is supposed to serve as a temperature regulating mechanism by causing blood flow in response to certain stimuli. These bodies are located in the subcutaneous layer of the skin, and range from ten to five hundred per square centimeter. They are most numerous in the nail beds, the tips of the palmar surfaces of the digits, and the soles of the feet.

Clinically, glomus tumors are subcutaneous nodules which, when given such stimuli as cold or trauma, precipitate severe paroxysms o pain. When located sublingually they may cause elevation or deformity of the nail.

Erosion of the bone of the distal phalanx with a smooth concave defect is shown to be typical roentgen picture of glomus tumor.

Treatment is simple excision of benign tumor.—*Richard E. Kinzer, M.D.*

Costero, J. Some problems related to the origin and meaning of pituitary gland tumors. *Arch. Path.*, Sept., 1948, *46*, 243–259.

Based upon a study of 150 tumors of the pituitary gland this paper emphasizes the multipotentiality of the pituitary anlage and discusses the proper classification of various pituitary tumors on the basis of their morphology and their probable relationship to the cell types of the embryonic pituitary.

The author feels that "oligochromic adenomas" are acidophilic adenomas which have lost some of their power to elaborate specific granules. He considers the so-called pregnancy adenomas of Poindecker and transitional adenomas of Kraus to be intermediate between acidophilic and oligochromic adenomas.

The various morphologic types of chromophobe adenomas are described and illustrated. The author considers them to be a true group with a common origin, attributing the great variation in their histopathological appearance to the multipotentiality of the cells. He feels that suprasellar cysts are simply persistent embryonic vesicles and proposes the name "archeoblastoma." Pituitary adamantinomas are rare and only those epithelial tumors producing calcifiable hyaline substance should be considered true adamantinomas.

Costero feels that true teratomas are also rare in the pituitary and that most so-called

teratomas show predominance of one structure and should be classified as hamartoma or choristoma.—*David D. Beiler, M.D.*

EVANS, W. G., and PICCIOTO, G. Chromophobe adenoma of the pituitary. *Brit. J. Radiol.*, July, 1948, *21*, 330–336.

Except for the few cases when a chromophobe adenoma is suprasellar, the first symptoms are caused by pressure on the pituitary itself: amenorrhea in the female and loss of libido in the male. Headaches are variable, but a constant finding at some time or other in the course of the disease is sellar erosion. Almost all patients suffer from pressure on the optic chiasm. Hemianopsia and scotoma are frequent: blindness or normal vision is rare. The course of the disease is usually progressive, with possible periods of arrest, and hypophyseal cachexia or blindness frequently results.

Surgical treatment can improve visual symptoms in 60 per cent and arrest their progress in most of the remainder, but the endocrine abnormality is usually permanent. Postoperative irradiation apparently improves the five year results.

One advantage of surgical treatment is the fact that it permits a histopathological diagnosis. When roentgen radiation alone is used, the following conditions must be excluded as far as possible: tumors at a distance from the sella turcica, craniopharyngiomas, intracranial aneurysms, meningiomas of the tuberculum sellae, malignant nasopharyngeal tumors with involvement of the basisphenoid, primary and metastatic tumors of the pituitary, and glioma of the optic chiasm. In the overwhelming majority of cases an accurate diagnosis can be made without biopsy.

Radiotherapy can be effective, but it must be given in sufficient doses. It is not effective in tumors which have become cystic; on the other hand, if improvement in the perimetric examination does not occur, the therapy has not led to a significant delay. Roentgen therapy usually produces no undesirable secondary effects, even with adequate dosage.—*E. F. Lang, M.D.*

NECK AND CHEST

SIMPSON, J. F., and HILTON, GWEN. Malignant disease of the pharynx, excluding the nasopharynx. *Proc. Roy. Soc. Med.*, July, 1948, *41*, 443–448.

Simpson cites two series of laryngopharyngeal cancers reporting five year survival rates of 4 and 5 per cent. Symptoms usually were insidious and most patients presented themselves in an advanced stage.

Histopathology, site of occurrence and operability determine mode of therapy which must be individualized for each case. Cancers involving the vallecula, epiglottis, and aryepiglottic fold respond well to irradiation. Growths involving the pharyngeal wall, pyriform fossa and the post-cricoid region are less susceptible to irradiation and should be resected if feasible. Any carcinoma thought to have invaded the laryngeal cartilage should be resected if possible.

Hilton reports 88 pharyngeal growths. The average duration of symptoms was six months before treatment. The greatest age incidence was 50–80 years with more males presenting. A surgical-radiotherapy team saw each case to determine therapy. All inoperable cases and lymphomas were treated by irradiation only. Local sepsis must be controlled before beginning irradiation. Seventy-one cases received full courses of irradiation; 15 are now alive with an average duration of life of 36.6 months. The others survived an average of 18.4 months.—*Donald R. Bernhardt, M.D.*

CLARK, J. V. A case of submucous abscess of the oesophagus. *J. Laryng. & Otol.*, July, 1948, *62*, 461–464.

A twenty-four year old British soldier in India developed a sore throat which was diagnosed as tonsillitis. This subsided in three days, but the patient then complained of severe dysphagia, being unable to swallow solids at all. A barium swallow demonstrated a typical benign stricture which did not relax even after a week of intensive belladonna therapy. The stricture involved the lower third of the esophagus. Esophagoscopy demonstrated a thick yellow membrane in this area. When this was peeled out 15 cc. of pus drained from the area. It was thought that the abscess was within the walls of the esophagus.

A true stricture then developed which was dilated in the usual manner, and the patient sent home with a size 14 bougie to be used regularly. A barium swallow showed the esophagus to be normal at this time with normal peristaltic waves in the involved area.—*F. M. Windrow, M.D.*

Lohman, A. J.M. Mediastinitis anterior chronica. *Acta med. Scandinav.*, Jan., 1948, *131*, 51–65.

Five cases of precordial complaints are described in which a chronic inflammation of the mediastinum (anterior) with an extensive adhesive formation of the visceral pericardium at the back of the sternum and xyphoid must be considered as the cause of the complaints. The origin is probably connected with a preceding acute tracheobronchitis or pharyngitis. The infecting organism was thought to be a streptococccus or influenza bacillus. The retrograde transport of the organism along the lymph nodes and lymph vessels of the anterior mediastinum is considered the most likely mode of infection. Surgical intervention is the treatment of choice for some cases. One patient was treated by resection of the lower part of the sternum and the xiphoid process. In roentgen diagnosis the lateral film of the chest is of great value. Tomography and kymography may be of value at times.—*Charles Nice, M.D.*

Fleischner, Felix G., Bernstein, Charles, and Levine, Bernard E. Retrosternal infiltration in malignant lymphoma. *Radiology*, Sept., 1948, *51*, 350–358.

In the authors' cases of intrathoracic malignant lymphoma, they have occasionally observed retrosternal infiltration, occurring alone or in conjunction with one or several of the usually noted localizations of lymphoma, but not previously described as such.

In a straight lateral view of the chest they distinguish two types of retrosternal infiltration:

(1) A soft tissue mass of even width, 0.5 to 2.0 cm. thick, extending from the level of the diaphragm to the level of the sternoclavicular joint, where it merges with normal soft tissue structures. It apparently represents a lymphomatous mass.

(2) A pad-like mass or several masses giving a lobulated appearance, of similar thickness, convex posteriorly with the base toward the sternum. Possibly this is the early stage of type 1.

Presternal edema was frequently associated, and in one case was the factor leading to search for retrosternal involvement.

The differential diagnosis includes accumulation of fatty tissue, pleural thickening, and inflammatory or neoplastic disease originating in the anterior chest wall structures, whether soft tissue or skeletal, primary or metastatic.—*Arthur A. Brewer, M.D.*

Kerley, Peter. Chronic diseases of the pericardium. *Proc. Roy. Soc. Med.*, July, 1948, *41*, 437–442.

The roentgen findings in gross pericardial disease are irregularity of the cardiac border, diminished or absent pulsation, limited diaphragmatic movements and associated pleural thickening. Pericardial adhesions most often affect the right heart border and the anterior surface. The most consistent aberration of cardiac silhouette in constrictive pericarditis is a continuously convex right border from diaphragm to aorta. Other less frequent contours are described. Usually the heart is central in position and is small although it may appear enlarged. The retrosternal and retrocardiac spaces may be obliterated and fixation of the heart may be noted, particularly during forced respiration in the lateral recumbent position. Pulsation may be increased or diminished in amplitude. Pericardial calcification is diagnostic of pericardial disease.

Kymographic studies are often diagnostic. Routine examination should include postero-anterior and lateral films taken in the recumbent position.—*Donald R. Bernhardt, M.D.*

Pulcherio, J. B., Jr. Estenose mitral. (Mitral stenosis.) *Brasil méd-cir.*, March, 1948, *10*, 151–164.

The author describes in detail the roentgen appearance of mitral stenosis and its complications. Kymographic studies are added in some cases and the roentgenograms are supplemented by line drawings.—*Ernst Schmidt, M.D.*

Mortensen, Vagn, and Warburg, Erik. Chronic constrictive pericarditis. *Acta med. Scandinav.*, March, 1948, *131*, 203–225.

A brief survey is given of the literature on chronic constrictive pericarditis, together with an account of a patient material comprising 25 clear-cut and typical cases besides 8 uncertain or complicated cases.

Clinical symptoms and electrocardiographic changes are discussed. Dyspnea, increasing circumference of the abdomen and tendency to edema were noted most frequently. Venous pressure was usually increased. The liver was consistently enlarged. A protodiastolic rhythm

(pericardial click) was often present. Electrocardiographic changes include flattening or inversion of the T waves in the limb leads.

On roentgenography the heart was of normal size in 18 cases, slightly enlarged in 2 and considerably enlarged in 5. In 12 patients areas of calcification were noted in the pericardium. Enlargement of the heart, pericardial calcification and perpetual arrhythmia tend to accompany each other. Kymography was performed in 18 cases and showed invariably small deflections or none at all. In 7 cases kymography was performed after operation, and in 3 of these cases there was no change in the picture, while in 2 cases the deflection became larger and in 2 cases the kymogram became normal.

Concerning etiology it is doubtful whether rheumatic infection plays any particular role. Tuberculosis, coronary occlusion and trauma apparently are etiological agents although, on the whole, we know very little about the etiology.

In cardiac insufficiency with pronounced edema in relatively young persons, in whom no diagnosis of the heart lesion seems obvious, we should always keep in mind the possibility of chronic constrictive pericarditis.—*Charles M. Nice, M.D.*

POTTER, BENJAMIN P., and GERBER, ISADORE E. Acute diffuse interstitial fibrosis of the lungs. *Arch. Int. Med.*, August, 1948, *82*, 113–124.

Both clinicians and pathologists encounter cases of diffuse pulmonary fibrosis of undetermined origin. Generally, these cases are characterized by a protracted clinical course without distinctive features. The postmortem examination usually reveals a non-specific chronic inflammatory process with extensive organization of the alveolar structures.

The paucity of pulmonary signs on physical examination is striking in view of the subsequent anatomic findings. However, this may be explained by the fact that the lesion is predominantly interstitial in location. A fairly constant finding is that of harsh breath sounds. The presence of crackling and moist rales, together with intensified or distant breath sounds, may be due to the focal bronchopneumonia present in some instances in association with the interstitial fibrosis. The disparity between the physical findings and the roentgenographic appearance is not surprising since similar features have been noted in so-called atypical or virus pneumonia, a disease in which the anatomic lesion is also predominantly in the interstitial tissue, usually about the bronchi.

The term acute diffuse interstitial fibrosis of the lungs was introduced by Hamman and Rich to describe a comparatively rare group of cases characterized by an acute clinical course and terminating fatally. The predominating symptoms are those of progressive dyspnea, cyanosis and a harassing non-productive cough, with death in respiratory failure or in failure of the right side of the heart. The pathologic features are those of diffuse fibrosis of the alveolar walls, with little involvement of the alveolar lumens.—*E. J. McDonald, M.D.*

BARDEN, ROBERT P., and COOPER, DAVID A. The roentgen appearance of the chest in diseases affecting the peripheral vascular system of the lungs. *Radiology*, July, 1948, *51*, 44–57.

This very interesting article describes the roentgen appearance of the chest in diseases which involve the peripheral vascular system of the lungs with reference to those conditions which produce an increased vascular permeability. In the field of allergy and pulmonary pathology, investigators have demonstrated the relationship of hypersensitivity states to pathologic changes in peripheral vessels. Rabbits sensitized to horse serum were injected with small amounts of antigen. Lesions were produced throughout the animal which were identical with the pathological lesions in patients dying with acute rheumatic fever, periarteritis nodosa, and serum sickness. The essential changes were widespread areas of focal necrosis in small arterioles and capillaries accompanied by a perivascular inflammatory reaction which may go on to healing with characteristic scar tissue.

Since a variety of clinical conditions may produce similar pathologic changes in peripheral blood vessels through the body, it is reasonable to assume that the changes in the lungs may reflect the entire pathological picture of the disease. The following disease conditions are considered:

Trauma. A direct blow or an explosive blast may by actual rupture or change of tone of the capillary walls cause increased vascular permeability. With the former the roentgenogram may show a localized dense lobar con-

solidation, while with a blast injury, patchy shadows over both lung fields are seen, because the entire capillary bed of the lungs may be affected. A careful clinical history and the rapid disappearance of the lung changes tend to differentiate this lesion from an infectious one.

Epidemic Influenza. An actual anatomic change in the lung capillaries is absent, but there is an increased permeability, probably due to some toxin elaborated by the organism. Massive edema and hemorrhage gradually increasing in severity produce a peripheral haze on the chest roentgengram which gradually moves toward the hila, and becomes confluent until the entire lung is obscured.

Disseminated Lupus Erythematosus. The changes in the chest roentgenograms may be similar to those of epidemic influenza. The changes are produced by necrotizing arteritis involving the arterioles of most organs of the body.

Acute Rheumatic Fever. This is primarily a collagenous degeneration and polyarteritis which involves many organs other than the heart. The term "rheumatic pneumonitis" to describe the roentgen changes in acute rheumatic fever is a useful one. A diffuse haze involves the middle and upper portions of the lung fields in a rather symmetrical manner. This appearance often occurs without evidence of heart failure, and is not associated with enlargement of the hilar shadows.

Exfoliative Dermatitis. This disease caused by many toxic agents may produce pathologic changes in other organs beside the skin. Roentgenograms of the chest during the acute phase may show a diffuse haze over both lung fields due to edema. The nature of the pulmonary shadows and the uniform diffuse symmetrical character suggest a focal perivascular origin.

Acute Glomerulonephritis. In this condition transient pleural effusions and pulmonary changes may be observed on the chest roentgenogram. It is probable that the same agent which causes the increased permeability of the subcutaneous peripheral vessels, accounting for the visible edema, may affect the pulmonary vessels as well. Again, as in acute rheumatic fever, without evidence of cardiac failure, passive congestion cannot be responsible for the chest findings.

Periarteritis Nodosa. This disease may coexist with or be indistinguishable from any of the previous conditions mentioned. The histopathologic lesion is a focal arteritis and may involve vessels in any organ. The small pulmonary vessels are thought to be involved in about 30 per cent of cases. Most of the patients will show pulmonary changes roentgenographically. In the acute stage of the disease, the roentgen shadows are massive and symmetrical, extending from the hilum to the middle lung zone. These changes may completely disappear in forty-eight hours if anti-allergic drugs are administered. Changes in the permeability of the vessel walls are considered the cause for the sudden onset and rapid clearing of the lung picture. In the chronic form of periarteritis nodosa the roentgen findings suggest bronchopneumonia, but the small patchy shadows migrate throughout the lung fields.

"Loeffler's Syndrome." This syndrome has been considered a manifestation of allergy and has been associated with migratory pulmonary shadows on the chest roentgenogram, and an eosinophilia in the blood. The roentgen appearance suggests a chronic symmetrical bronchopneumonia, but is not characteristic of pulmonary vascular disease.

The authors have presented a logical approach to the correlation of the roentgen appearance of the chest in hypersensitivity states with the basic pathologic changes which have affected the peripheral vascular system.—*George P. Keefer, M.D.*

Potts, Willis J., and Gibson, Stanley. Aortic pulmonary anastomosis in congenital pulmonary stenosis. *J.A.M.A.*, May 22, 1948, *137*, 343–351.

In November, 1946, Potts, Smith and Gibson reported a new surgical procedure for the relief of congenital pulmonary stenosis. The operation consisted of a direct anastomosis between the aorta and the pulmonary artery. The use of the aorta was made possible by the invention of a clamp which permits blood to flow through this vessel while the anastomosis is being performed. The operation differs from the Blalock and Taussig method only in the fact that the aorta itself rather than one of its branches is used to direct additional blood to the lungs.

This is a report on the authors' experience with this operation in 45 patients. An additional 7 patients were explored but operation could not be accomplished.

The article reiterates the criterion for diagnosis of tetralogy of Fallot and the variations from the classical physical findings and symptoms. It is pointed out that the roentgenological examination is probably the most important single feature of the examination. A roentgenogram in the posteroanterior position almost always reveals a heart that is normal in size or only slightly enlarged. Gross cardiac enlargement militates strongly against the diagnosis of tetralogy of Fallot. The shape of the heart is subject to considerable variation. It is gratifying if the film shows a heart that is boot shaped, appearing to ride high on the diaphragm, lying more of less transversely in the chest. However, the heart contour may be normal. A slight concavity is usually present in the pulmonary area. Prominence in the pulmonary conus area indicates a large pulmonary artery and should immediately cast doubt on the diagnosis of tetralogy of Fallot. The appearance of the hilar regions must also be noted. If there is evidence of increased vascularity, it is an important sign that the lungs are receiving a sufficient amount of blood. Fluoroscopic examination is important in determining the amount of blood flow through the pulmonary arteries. With the eyes thoroughly accommodated search should be made for expansile pulsations in the hilar regions. Absence of pulsation is the rule in pulmonary stenosis. Fluoroscopic examination in the left anterior oblique is obligatory. In this position one is able to determine whether there is an abnormally clear area below the aortic arch; in other words, whether the pulmonary arteries are carrying less blood than normal. An abnormally clear pulmonary window is one of the most reliable signs of pulmonary stenosis. One should be very sure that the patient does not also have a right-sided aorta, as it is very important to the surgeon as to what side he attempts to make his anastomosis. Approximately 15 to 20 per cent of cases the aorta descends on the right. In most instances this is easily determined with a barium swallow.

In 4 of the patients coming to operation, the clinical diagnosis was tricuspid atresia. The physical signs are identical with those encountered in tetralogy of Fallot. There are two distinguishing characteristics, however. The roentgen silhouette in the anteroposterior view shows a gentle rounding of the left cardiac border due to hypertrophy of the left ventricle and an absence of the normal fullness in the region of the right ventricle. The second diagnostic feature is left axis deviation in the electrocardiogram.

The mortality of the operation on the 45 patients in whom the anastomosis was performed was 8.8 per cent. Most of these patients prior to operation were severely crippled and of the 41 who survived the surgery, 39 have been markedly improved. It has been only a year since the first child was operated upon when this report was made, so it is not possible to determine the ultimate prognosis in this group of cases.—*E. A. Addington, M.D.*

MACLEAN, K. S. The relation of idiopathic pleurisy and pleural effusion to tuberculosis. *Guy's Hosp. Rep.*, 1948, *97*, 133–184.

This paper is based on the author's thesis approved for the degree of M.D. (Cambridge). It includes: a review of early observations and views on pleurisy; the change in the medical profession's attitude brought about by Koch's discovery; a tabulation of,—and discussion of the discrepancies in,—the more important follow-up studies of patients witn pleural effusion; an extensive list of references; and a report of Maclean's study of the records of 183 patients with pleural effusion and 126 patients with dry pleurisy who had been admitted to the three main Royal Navy Hospitals in England during 1940 and 1941, and who had been followed for three years or more.

From the last come these conclusions:

(1) Many instances of dry pleurisy (as opposed to pleural effusion) are due to causes other than tuberculosis. The tuberculous morbidity in the three to four years after return to duty following pleural effusion was 15.9 per cent; following dry pleurisy, it was 1.9 per cent. Considering all factors, the tuberculous morbidity in a five year period following pleural effusion in the Royal Navy is approximately 25 per cent. However, the history of a pleuritic incident six months before the development of fluid was surprisingly frequent in the analysis of patients with pleural effusion. This sequence of events seems strong evidence of a tuberculous etiology and is presumably due to the different response of the pleura before and after tuberculin hypersensitivity has developed. Therefore, every patient with unexplained dry pleurisy should be carefully observed; if no further evidence of tuberculosis occurs in nine months, the likelihood of subsequent infection is slight.

(2) Pleural fluid complicates obvious pulmonary tuberculosis "much less frequently than might be expected." Miliary tuberculosis is infrequently accompanied by an effusion. From other authors' studies, Maclean concludes that many adults develop wet pleurisy some months after Mantoux conversion.

(3) Subsequent tuberculosis is more likely to occur when the effusion is large, *its duration is long*—for "several months" (the most important single prognostic factor, by far), pyrexia is sustained, and (less definitely) if the patient fall in the 20–30 year age group.

(4) The commonest site for tuberculosis to develop following pleural effusion is the upper part of the lung fields, usually on the same side as the effusion. About two-thirds of relapses occur within two years.

(5) Probably the commonest cause of pleural effusion in patients over forty is malignancy. This fluid is far more often bloodstained than is a tuberculous effusion. An effusion in a patient suffering from bronchial neoplasm, although of grave prognostic significance, does not necessarily imply that the case is beyond surgical aid.—*Henry G. Moehring, M.D.*

Goodrich, William A. Pulmonary edema. *Radiology*, July, 1948, *51*, 58–65.

The physiological factors which produce pulmonary edema are reviewed. These are (1) increased intracapillary hydrostatic pressure; (2) decreased osmotic pressure of the blood; (3) increased capillary permeability.

The variation in these basic physiological factors caused by cardiac failure, nephritis, excessive administration of parenteral fluid, and the mechanism of production and location of pulmonary edema as observed on the chest film in each condition are described in some detail.

To differentiate the shadows produced by pulmonary edema and in chest films from other conditions, the author lists the following observations: (1) the diffuseness of the shadows; (2) relative clearness of the extreme apical and basal lung fields; (3) a tendency for the shadows to vary in size and to show a shift in location on serial films; (4) enlargement of the vascular shadows; (5) enlargement of the heart.

In cardiac failure, where pulmonary edema results from increased hydrostatic pressure and increased capillary permeability, pleural effusion is more commonly seen with chronic failure. In acute failure, the edema tends to occur in the central portion of the lungs, the apices and bases are clear and pleural effusion is seldom seen.

Nephritis may cause pulmonary edema by variations in one or all of the three basic physiologic factors, i.e.: a low serum protein, plus low osmotic pressure in pure nephrotic nephritis; increased capillary permeability and increased hydrostatic pressure in acute glomernulonephritis.

With the excessive administration of parenteral fluids, hydrostatic pressure increases, serum proteins are decreased, and osmotic pressure of the blood is lowered. Cardiac enlargement and pleural effusion may accompany the pulmonary edema.—*George P. Keefer, M.D.*

ABDOMEN

Greenwood, Frank, and Samuel, Eric. The pathological gallbladder. *Brit. J. Radiol.*, Sept., 1948, *21*, 438–445.

The authors, from Johannesburg, South Africa, have analyzed 500 consecutive cholecystographic examinations. When the gallbladder was not visualized after one dose of the opaque medium, or was poorly visualized (i.e., not seen fluoroscopically), two further doses were administered at twenty-four and forty-eight hour intervals. Films were made in prone and erect positions with and without compression and if necessary tomography was resorted to. Three hundred and sixty-two were finally regarded as normal, but of these 36 had to have the extra two doses for demonstration of this fact. In other words, 1 out of 10 normal gallbladders requires the three dose technique.

Of the abnormal 138 cases, 32 per cent showed no function and no stones were visible. Many of these did not submit to surgery, so the number with stones could not be determined Of the remaining 94, one-third had opaque stones which could be seen without cholecystography, and two-thirds showed nonopaque stones.

Eighteen per cent of these 94 showed stones which could be seen only in the erect position, and it is the authors' opinion that routine fluoroscopy of the gallbladder in the erect position, using serial roentgenography and compression, is essential in every examination of the gallbladder. Tomography is valuable if the gallbladder cannot be separated from overlying intestinal gas.—*E. F. Lang, M.D.*

HUGHES, C. R., HANNAN, J. R., and MULVEY, B. E. Cholangiography in stone stricture and operative injury of biliary ducts. *J.A.M.A.*, June 19, 1948, *137*, 687–690.

The authors review their experience in 90 consecutive patients who have had exploration of the common duct for stone, stricture or stenosis of the biliary duct.

The indications for cholangiography include all patients showing jaundice or a history of jaundice; if multiple small stones are found in the gallbladder at operation; if the ducts are dilated or obviously diseased; if a stone in the duct is suspected on palpation; if a small fibrotic gallbladder is discovered; if bile aspirated from the duct is muddy; if the liver is cirrhotic.

The authors use 35 per cent iodopyracet solution. The dye is observed under fluoroscopic visualization as injected. Spot films are taken at the time of injection and at fifteen and thirty minutes following the injection. Postoperative cholangiography was 93.3 per cent accurate in demonstrating common duct stones not found at previous surgical exploration. The examination is done at the time of operation and repeated later before removal of the T tube. Cholangiograms following operation for stricture are of value to show the rate of entrance of bile through the anastomosis into the duodenum.—*V. L. Peterson, M.D.*

GOIN, LOWELL S. Fibrocystic disease of the pancreas. *Radiology*, July, 1948, *51*, 36–43.

The author reviews in some detail the clinical, pathologic and roentgen findings observed in fibrocystic disease of the pancreas. While this disease was considered rather rare, autopsies indicate that it accounts for about 4 per cent of infant deaths. The disease is probably of congenital origin, since it occurs frequently in the newborn and in more than one child in a family. It is characterized by repeated respiratory infections; signs and symptoms of celiac-like disease. The cause of the disease is unknown, but an obstruction of the small pancreatic ducts and subsequent dilatation of the acini is very likely a basic factor. Most observers believe that a vitamin A deficiency caused by absent or inadequate pancreatic enzymes produces the lowered resistance of the lung to infection, and the resulting pulmonary picture.

Histopathologic examination of the pancreas shows atrophy of the acinar parenchyma; interacinar and interlobular connective tissue is increased. The lungs are the site of suppurative bronchitis and bronchiectasis. Bronchopneumonic patches are scattered through the pulmonary tissue.

These lung changes account for the bilateral hilar prominence, linear densities, bronchopneumonic patches, and occasional localized areas of emphysema seen on the roentgenogram of the chest. These findings should make us suspect the presence of pancreatic fibrocystic disease, especially when associated with gastrointestinal symptoms of celiac-like disease.

Children with this disease fall into three groups:

1. Those who die in the first week or two with meconium ileus, intestinal stenosis or atresia.
2. Those who, usually in their first year, display gastrointestinal and respiratory tract findings, the presenting symptoms usually being those of pulmonary infection. This group has repeated attacks of cyanosis and die of respiratory infection.
3. Those who present signs and symptoms of celiac-like disease—i.e., failure of weight gain, foul and foamy stools, intolerance to fats and carbohydrates, associated with bronchitis and bronchiectasis.

If the disease is recognized early, adequate treatment can be instituted and some babies may be saved. Therapy is directed to the control of the pulmonary tract infection by chemotherapy and antibiotics. Establishment of adequate diet is obtained by supplemental feeding of the pancreatic enzymes, casein hydrolyzates, etc.—*George P. Keefer, M.D.*

SELMAN, JOSEPH, and BENDER, JOHN R. Mesenteric lipoma in a child. *Radiology*, July, 1948, *51*, 66–70.

The literature dealing with lipomas and other fatty tumors is reviewed. The paucity of reported cases of intra-abdominal lipoma is noted.

The authors report a single case of mesenteric lipoma in which a preoperative roentgen diagnosis was made. The report is illustrated with reproductions of two roentgenograms. The reproductions are of sufficient quality to demonstrate the *radiolucency* of the tumor.—*Richard E. Kinzer, M.D.*

Likely, Stanley David, Lisa, James R., Stitch, Melvin H., and Stein, H. D. Primary malignant disease of the small bowel. *Arch. Int. Med.*, August, 1948, *82*, 206–216.

Seventeen cases of malignant growth of the small bowel are reported, and the recent literature is reviewed. They are of uncommon occurrence. Certain clinical features suggest the possibility of their presence. Intermittent obstruction is a common manifestation. In some cases the presenting symptom is mild unexplained nausea. In the presence of anemia of gastrointestinal origin for which no adequate explanation is found on examination of the stomach and large bowel, the small bowel should be thoroughly investigated. The prognosis is usually considered poor in cases of carcinoma and lymphosarcoma and good in cases of argentaffinoma. The more recent literature suggests that the prognosis may depend largely on early diagnosis rather than on the type of tumor, particularly in reference to carcinoma and lymphosarcoma. Roentgenologic studies are most valuable. The therapy is radical resection. Awareness of the possibility of the existence of the condition remains the prime necessity for early diagnosis.—*E. J. McDonald, M.D.*

Wells, Josephine. Mucosal pattern of the terminal ileum in children. *Radiology*, Sept., 1948, *51*, 305–309.

While the normal mucosal pattern of the barium-filled terminal ileum in the adult on pressure appears as slender regular lines produced by the mucosal folds, that in children is found to present a different pattern. Here there are noted small rounded filling defects giving a cobble-stone appearance. The writer demonstrated this in 14 cases of children having no history of gastrointestinal symptoms. Another child who died of a non-relevant condition was autopsied and the lymph node collections were numerous in the terminal ileum. A barium sulfate suspension was smeared on the surface and the typical cobble-stone appearance was demonstrated on roentgen examination. The writer concludes that this appearance is normal in children and is due to the lymphoid tissue which is more marked than in adults. Occasionally in young adults having an inflammatory lesion in the ileum with local lymphadenitis the cobble-stone appearance may also be noted.—*J. Paul Bennett, M.D.*

Pendergrass, Robert C. Extrinsic deformities of the colon. *Radiology*, Sept., 1948, *51*, 320–325.

The difficulty of differentiation of extrinsic and intrinsic defects of the colon is pointed out. No hard and fast rules can be drawn. Even after correlation of the history, physical findings, roentgenologic data and knowledge of the anatomy and pathology, one cannot honestly state whether a defect is intrinsic or extrinsic.

Extrinsic deformities of the colon may be roughly grouped as follows: (1) deformities from enlarged viscera, including tumors of these viscera; (2) deformities from inflammatory processes, adhesions, and endometriosis; (3) deformities from retroperitoneal tumors, mesenteric tumors, and omental tumors; (4) deformities from tumors of the colonic wall not invading the mucosa; (5) deformity from intra-abdominal and inguinal hernia.

The author has found it very helpful, in certain instances, to make a vaginal examination under fluoroscopic observation while the colon is filled with barium. This procedure helps to separate masses in the right pelvis from the distended cecum. On occasion, a mass thought to be an ovarian cyst has been demonstrated to be a distended pelvic cecum.—*Chauncey N. Borman, M.D.*

Bell, Joseph C., and Douglas, James B. Roentgen-ray diagnosis of malignant and potentially malignant lesions of the colon and rectum. *Radiology*, Sept., 1948, *51*, 297–304.

Because of public enlightenment patients are now seeing their physicians earlier in cases of cancer. Malignancies of the colon are consequently encountered by the physician at an earlier stage, at which diagnosis is more difficult. Seventeen per cent of all deaths caused by cancer are due to cancer of the colon or rectum. The radiologist should obtain detailed history concerning bowel habits, abdominal discomfort or pain interfering with passage of material, rectal bleeding, indigestion and evidence of progressive anemia, if such history is not supplied. Sigmoidoscopy is important in the study of the rectum and sigmoid; some lesions not demonstrable by roentgen ray may be so demonstrated. The use of sigmoidoscopy in conjunction with the roentgen study should therefore be advised by the roentgenologist. Cleansing of the bowel thoroughly both before sigmoidoscopy and the barium study is of great

importance. The authors use castor oil for this purpose.

The writers make the examinations of the colon in the right and left oblique projections, the angles being determined by the fluoroscopic findings. A true lateral projection of the filled rectum and rectosigmoid region is also obtained after which a film of the entire colon is made in the posteroanterior projection after filling and also after evacuation. The writers have a special three-contact quick change-over switch in parallel with the change-over switching apparatus in the spot film tunnel permitting an exact duplication on the film of any roentgenoscopic image by placing the cassette over the area of interest. Serial exposures aid in differentiating stricture due to diverticulitis from a cancer. If there is any question concerning the nature and significance of a filling defect it is essential that re-examination be made at the earliest possible time.

Double contrast examinations are of value, especially in demonstrating polyps. The routine barium enema will seldom demonstrate these. The writers state that the ordinary barium enema study and the double contrast require such different techniques as to necessitate separate examinations rather than a combination of the two. They use twice as heavy a barium sulfate concentration for the contrast study as for the ordinary barium enema. Stereoscopic films are made in the prone and right posteroanterior oblique positions before expulsion of the air. As polyps are acknowledged to be potentially malignant, the double contrast study is of importance. For the demonstration of polyps in the rectum and sigmoid, sigmoidoscopy is the most accurate method. There should be close cooperation between the sigmoidoscopist and the radiologist. Even when proctoscopy reveals a low polyp the barium study should be made as additional polyps may be found at a higher level.

An examination of a large number of cases of cancer of the colon and rectum revealed that slightly over 50 per cent of the lesions proved to be operable and free of demonstrable cancer after five years. If the diagnosis is made before there is lymph node involvement, these results can be improved by probably 10 per cent.—*J. Paul Bennett, M.D.*

SKELETAL SYSTEM

PETERMAN, M. G., FREY, W. B., and KASTER, J. D. Value of routine roentgenograms of the wrist in a pediatric service. *Am. J. Dis. Child.*, May, 1948, *75*, 671–687.

This paper is a report of the investigation of 684 infants and children for evidence of a pathologic process by means of routine roentgenograms of the wrists. For means of determining a delay in the centers of ossification the revised Leonard "ossification index" was used exclusively. A number of pathologic findings were visualized which were consistent with the following conditions: rickets, hypothyroidism (delayed ossification), cerebral birth injury (delayed ossification), mental retardation (delayed ossification), fibrocystic disease of the pancreas (delayed ossification), pituitary dwarfism (delayed ossification), cerebral dysgenesis with hypothyroidism (delayed ossification), delayed ossification (cause undetermined), scurvy, syphilis, lead intoxication, mongoloidism, osteochondromatosis, chondrodystrophy, pubertas precox, and osseous changes not attributable to any definite clinical entity.

Two series were done independently, one in a private pediatric service, and the other in a charity hospital pediatric service. Of 333 hospital cases 34, or 10.21 per cent, and of 351 private patients studied 35, or 9.97 per cent, of the roentgenograms of the wrists revealed evidence of a pathologic process.—*R. S. Bromer, M.D.*

STEINBERG, CHARLES LEROY. Brucellosis as a cause of sacro-iliac arthritis. *J.A.M.A.*, Sept. 4, 1948, *138*, 15–19.

The author gives a short introductory general discussion of brucellosis followed by an analysis of other writers on the subject, particularly as it pertains to joint pains. A serologically but not bacteriologically proved case report is given. In addition to the usual other symptoms the patient complained of pain in the hip. This was shown to be due to well defined areas of destructive arthritis in the sacroiliac synchondroses.

The question is raised as to whether rheumatoid arthritis is due to brucellosis. After a study of 20 cases of rheumatoid spondylitis, the writer's answer is no.—*Allan Tuggle, M.D.*

MARÓTTOLI, OSCAR R. Concepto general de seudoartrosis. Consideraciones estadisticas. (A general concept of pseudarthrosis; statistical considerations.) *An. de cir.*, March, 1948, *13*, 23–32.

The author discusses the North American

literature on non-union of fractures, covering both general and specific causes. He quotes several classifications of non-union, and recommends that advocated by Codivilla, in 1909, which seems most workable, comprising only delayed union and true non-union. True non-union is divided into cases with and without loss of bony substance. Those without loss of bone are further divided into recent cases with and without difficulty in immobilization, and older cases in which osteoplastic capacity is impaired or lost.

There is considerable discussion of changes in osteoplastic activity due to atrophy and eburnation of fractured ends, and due to soft tissue interposition.

A large series of statistics from Europe and South America is quoted; all, as in this country, show that non-union is most common in the navicular and the femoral neck.

The author quotes a series of over 7,000 personally treated patients with fractures of all bones, with only 8 cases of true non-union, a phenomenally low percentage of 0.001.

No mention is made of further surgical therapy in true non-union.—*William S. Wallace, M.D.*

Cassidy, William J., Allman, Francis C., and Keefe, Gerald J. Osteopetrosis. *Arch. Int. Med.*, August, 1948, *82*, 140–158.

Roentgenologically considered, osteopetrosis is a skeletal dystrophy in which increase in the density of the roentgenographic shadow cast by bone is the most characteristic feature. The usual architectural features of normal bone, such as fine trabeculations of the substantia spongiosa, well proportioned cortices and marrow cavities, careful modeling or tubulation and sharply contrasting densities, are missing. The building of osteopetrotic bone appears to have been sublet to an amateurish contractor, who had an abundance of mineral salts available and used them with a lavish, if untrained, hand. While the long bones, ribs and pelvis are the structures most commonly involved, the head, the spine and the bones of the hands and feet are frequently seen to manifest the changes of this disease.

While decidedly dense, osteopetrotic bone is not always uniformly so. Striking variations often occur in the shafts of the long bones, the carpal and tarsal bones and in the flaring portions of the iliac bones. At times translucent bands of decreased density of varying width are seen near the epiphyseal plate and often extending well up the diaphysis. These are transverse strata of defective loose substantia spongiosa. They represent a period of growth in which excessive destruction of cartilaginous lattice has occurred and are somewhat of a paradox in osteopetrosis. Certainly some interruption or temporary cessation of the usual course of events in the laying down of osteopetrotic bone has occurred.

The frequent association of osteopetrosis with osteomyelitis and delayed dentition are probably attributable to an impaired vascular supply, and consequently the nutritive requirements for normal development and maintenance of a healthy state are inadequate. Fractures, likewise, are a common observation in the patient with osteopetrosis. These usually heal with the production of adequate, and at times, abundant, callus. However, delayed union is not unusual. Evidence of healed or healing fractures has been discovered with the patient having been unaware of their occurrence

Ordinarily, the base of the skull is first and most decidedly affected by this disease. The bones of the calvarium, however, may also be involved and become so uniformly dense that no distinction can be made in the tables and the diploic portion. Obliteration and effacement of the mastoid cells and air spaces of the paranasal sinuses are fairly common. Narrowing of the various foramens of the skull, with compression of the corresponding emerging nerves, is not rare.

In the infant the outlook for life is generally poor, with death resulting from extreme inanition and intercurrent infection. Those who do survive usually have the stigmas of blindness, deafness, hydrocephalus, anemia and multiple fractures. In the adult, except for anemia and multiple fractures, the prognosis is generally favorable. Osteomyelitis is a serious complication. The treatment is ineffective. Several attempts have been made to mobilize calcium without success.

The authors' review of the entire subject is complete, well presented and substantiated by a voluminous bibliography.—*Eugene J. McDonald, M.D.*

Rathburn, J. C. Hypophosphatasia. *Am. J. Dis. Child.*, June, 1948, *75*, 822–831.

In this paper an unusual and previously unreported type of faulty bone development is

presented. A low to absent alkaline phosphatase appeared to be the primary defect responsible for the clinical picture. The patient was a boy, three weeks of age, born at full term following a normal seven hour labor. There was no familial history of bone diseases in two generations prior to this child. The roentgen examination showed marked decalcification throughout the bones of the body, membranous and cartilaginous. Decalcification was very striking in the skull, there being only a few patches of ossification present in the parietal and frontal regions. The base of the skull was moderately well calcified. The posterior arches of the lumbar vertebrae were indistinguishable from the soft tissue shadows, and the ribs of the thorax showed deformities and flaring of their ends. Some of most prominent areas showing lack of normal calcium density were seen at the metaphyseal ends of the long bones. Trabecular projections of non-osseous density, extending up into the diaphysis, were seen. The epiphyses appeared to be unaffected, except for a slight degree of osteoporosis and it would appear that the bony maturation, so far as the secondary epiphyseal centers at the knee were concerned, was within normal limits.

The alkaline phosphatase ranged during the patient's stay in the hospital from 0.4 to 0.0 and 0.15 Lowry-Bessey units, the normal range for children being 5.15 U. At postmortem, the long bones showed normal hyaline cartilage at the growing ends below the articular cartilage. There was a wide, irregular zone of proliferative cartilage with disorganized maturation of cartilage cells and no evidence of calcification of the intercellular matrix. The normal regular invasion of cartilage by capillaries was completely disrupted and as a result, long, irregular, cartilaginous processes projected into developing osteoid tissue. The osteoid tissue was deposited abundantly on the uncalcified cartilaginous intercellular substance and on the outside of the shaft but was almost completely uncalcified for some distance along the shaft. This accounted for fractures seen in a radius and an ulna. The picture resembled that of severe rickets. The vault of the skull showed a most unusual picture, the bony framework having been laid down perfectly in the form of osteoid tissue.

The membranous condition of the vault of the skull with the slightly elevated serum calcium and phosphorus suggested the possibility of osteogenesis imperfecta as the diagnosis. However, in osteogensis imperfecta, micromelia is secondary to fracture, the nose is of normal contour, the serum phosphatase is normal or increased, and the long bones are slender with thin cortices. Microscopically osteogenesis imperfecta usually shows a short zone of proliferative cartilage, whereas in the case reported, the proliferative cartilage was increased and was highly irregular. Rathburn also felt that achondroplasia and renal hyperparathyroidism could be excluded.

Treatment failed to change the course of the disease.—*R. S. Bromer, M.D.*

LAPIDUS, PAUL W., SLOBODY, LAWRENCE B., GERMANSKY, GEOFRED, and WILLNER, MILTON M. Eosinophilic granuloma of bone; report of three cases. *Am. J. Dis. Child.*, June, 1948, *75*, 900–909.

Three cases of eosinophilic granuloma of bone are reported which are added to 62 previously reported cases. The first patient had a lesion in the upper humeral shaft, the second in the sternum and the third in the roof of the acetabulum. The authors regard eosinophilic granuloma as a benign, probably inflammatory lesion of unknown origin which may be multiple or solitary. They regard the changes in the roentgenogram, although presenting certain diagnostic features, as not characteristic enough for any positive diagnosis. Usually round, ovoid or irregular, punched-out areas of varying size occur. The osteolytic lesions apparently originate in the medullary portion of the bone and have a fairly well outlined, occasionally sclerotic, margin. Since the lesion rapidly increases in size, it may cause cortical perforation or sometimes expansion of the cortex. Periosteal proliferation and cortical thickening also may simultaneously be produced with expansion of the lesion. Pathologic fractures of the weakened bone are frequently produced, followed by periosteal callus formation and rapid healing.—*R. S. Bromer, M.D.*

GENERAL

ARDRAN, G. M. Armillifer armillatus; a note on three cases of calcification of the cysts in man. *Brit. J. Radiol.*, July, 1948, *21*, 342–345.

Armillifer armillatus, an arthropod and one of the tongue worms or linguatulidae, lives as an adult in the bronchial tree of snakes, and its eggs can be transmitted to the gastrointestinal tracts of men who handle infested

snakes. The eggs develop into larvae or nymphs which eventually encyst in the peritoneum or in any of the organs of the celomic cavity. Only a small percentage of the nymphs calcify and by that time they are dead and not productive of symptoms. The calcifications are circular or C-shaped, with the band of calcification about 3–5 mm. in width, and the overall diameter of the cyst 3–4 cm.

Calcifications have been found in the lung, in the peritoneal cavity, on the peritoneum and in the liver. The calcifications are entirely typical and cannot be mistaken for other conditions if they are seen en face.

Infestation is known as porocephaly.—*E. F. Lang, M.D.*

Blanchard, A. J., and Boone, F. H. Reticuloendothelial granulomatosis; report of two cases of Hand-Schüller-Christian disease. *Am. J. Dis. Child.*, July, 1948, *76*, 1–13.

The authors present 2 cases of Hand-Schüller-Christian disease the features of which tend to support the hypothesis that the condition is a systemic reticulo-endothelial granuloma of unknown origin. They produce evidence to suggest that the presence of cholesterol in the lesion is a secondary consideration resulting from the ingestion of the lipid from the tissue fluids by the macrophages of the preformed granulomas.

They state that this theory fits the well recognized fact that the value for serum cholesterol is normal in most cases. With the onset of necrosis in the lesions, this ingested cholesterol is liberated from the cells and is observed lying free in the lesions in crystalline form. From these sites it may be absorbed into the blood stream and give rise to a transient hypercholesteremia.

Hand-Schüller-Christian disease has been grouped with Niemann-Pick disease and Gaucher's disease as a primary congenital lipodystrophy. In support of this theory, the hypercholesteremia of some of the cases and the xanthomatous nature of the lesions were cited. However, with the study of increasing numbers of cases, it was observed that the great majority did not show a significant increase in blood cholesterol and that in the various lesions encountered in a given case, foam cells were often a minor feature. The 2 cases reported tend to support this hypothesis. In the original descriptions of the disease, emphasis was laid particularly on the clinical and roentgenologic observations, rather than on the more fundamental anatomic bases. Gradually, with study of increasing numbers of cases it has been recognized that the clinical features in a given case depended solely on the localization of the xanthomatous deposits. Thus, the clinical conception of the condition became wider and wider. It was emphasized consistently that the histologic criterion necessary for anatomic diagnosis was the presence of foam cells in cholesterol-containing granulomas.

With this criterion in mind, a number of cases showing reticuloendothelial granulomas were described. These were considered by the authors as entities distinct from Hand-Schüller-Christian disease and excluded that diagnosis chiefly because of the absence of foam cells from the lesions. Among these conditions may be mentioned: (1) infectious reticuloendotheliosis, (2) Letterer-Siwe disease and (3) solitary or eosinophilic granuloma of bone. Wallgren considered that the infectious reticuloendotheliosis and Letterer-Siwe disease were essentially the same condition and classified them as non-lipid reticuloendotheliosis. The authors regard Wallgren's cases as a definite link between this group of cases and Hand-Schüller-Christian disease.

With regard to eosinophilic granuloma of bone, the authors quote Farber and Gross who suggested that the condition should be considered as exceptional examples of Hand-Schüller-Christian disease, with a single bony lesion and minimal visceral damage.

The authors' first case showed dissemination of reticuloendothelial granulomas which, although showing an occasional fatty droplet, failed to exhibit the characteristics of foam cells and cholesterol crystals. Tumor-like proliferation of the macrophages was particularly striking in the biopsy specimen from the skull, which showed a considerable number of mitotic figures.

In the second case the most pronounced changes were present in the lungs, skin and ribs and skull. The peculiar cutaneous infiltration is described in cases of Letterer-Siwe's disease. In the original biopsy specimen from the rib the histopathologic features were those of a non-lipid granuloma. At the autopsy several months later, sections from the same area showed extensive necrosis with acicular spaces representing cholesterol crystals and there was massive infiltration of foam cells. In the earlier stages the case presented features

described in Letterer-Siwe disease, but with full maturation of the lesions, the complete picture of Hand-Schüller-Christian disease was evident. This case seems to support Wallgren's contention that the two conditions are essentially the same and represent merely the early and the late stages of one process.—*R. S. Bromer, M.D.*

LINDSAY, STUART, REILLY, WILLIAM ANTHONY, GOTHAM, THELMA J., and SKAHEN, RICHARD. Gargoylism. II. Study of pathologic lesions and clinical review of twelve cases. *Am. J. Dis. Child.*, Sept., 1948, *76*, 239–306.

This paper follows a previous publication by Reilly and Lindsay which gave in detail the clinical findings in 18 cases of gargoylism. In this paper the authors present a complete study of 12 cases, 8 of which came to autopsy and 4 in which surgical pathological findings were obtained. They discuss carbohydrate storage in the disease and its physiologic aspects, and also the relation to carbohydrate and other macromolecular storage diseases.

The case histories of the 12 cases are given in detail. Then follow sections on the review of pathologic lesions of the condition recorded in the literature, the cellular lesions, histochemical studies, hereditary aspects, leukocytes, and carbohydrate metabolism in gargoylism.

The author's summary and conclusions are:

1. Gargoylism is a disease of early childhood, characterized by striking gross and microscopic alterations in almost all tissues of the body.

2. The basic lesion is the intracellular and extracellular deposition and storage of a substance giving the histochemical reactions for glycogen.

3. Certain histochemical data presented suggest that the glycogen may be combined with protein.

4. Laboratory studies indicate that glycogen storage process in gargoylism is not associated with demonstrable alteration in carbohydrate metabolism, as in true glycogenosis (von Gierke's disease).

5. Widespread involvement of most tissues, including the nervous, cardiovascular, reticuloendothelial, endocrine, skeletal and other systems, explains the protean clinical manifestations of the disease.

6. The lesions of gargoylism are similar to or identical with some of those of the other macromolecular storage diseases of both endogenous and exogenous origin.

7. Further, more precise, histochemical, chemical and enzymatic studies on the material stored in the tissues are indicated. Combination of glycogen with a protein stored in the cytoplasm of the cells may explain the inconsistent histochemical staining reactions recorded up to date.—*R. S. Bromer, M.D.*

ROENTGEN AND RADIUM THERAPY

GOLDMAN, R., EGEBERG, R. O., WARE, E. R., EVANS, E. R., and FISHKIN, B. G. Clinical experience with nitrogen mustard therapy. *Arch. Int. Med.*, August, 1948, *82*, 125–139.

Forty male patients who had Hodgkin's disease, lymphosarcoma, leukemia and related diseases were treated with bis-beta-chloroethyl-amine hydrochloride (nitrogen mustard). The therapeutic application of the drug results in a prompt response which is more rapid than that which occurs after roentgen therapy, but the remission produced is of much shorter duration.

Nitrogen mustard appears to offer promise of almost certain remission in Hodgkin's disease and polycythemia vera. Lymphosarcoma, reticulum cell sarcoma, chronic leukemia and mycosis fungoides respond in slightly more than half of the cases, but such a result cannot be predicted in advance. The response of the other diseases appears to be less consistent, and the group of patients described in this report is too small to permit conclusions of a general nature. The earlier the clinical state of the disease, the more likely is the remission to be clinically significant and the more possible the restoration of the patient to social and economic usefulness. For diseases in which nitrogen mustard has proved occasionally useful, one course may be tried to determine the patient's response. This should be done for the remission itself and to defer the development of resistance to roentgen rays. Since the first adequate course of nitrogen mustard, judged by the hematopoietic response, appears to be the most effective, failure to respond to the first course obviates the necessity of repeating treatment. Recently, several research groups have begun to study the effects of nitrogen mustard when combined with roentgen therapy. It is hoped that this will prolong the duration of the remissions. The rapid response to the drug complements the slower but more prolonged effects of roentgen irradiation, and this in itself is of value.—*Eugene J. McDonald, M.D.*

Snapper, I. Treatment of multiple myeloma with stilbamidine. *J.A.M.A.*, June 5, 1948, *137*, 513–516.

Thirty-five patients with multiple myeloma have been treated by intravenous or intramuscular injections of stilbamidine. The dose was 50 mg. the first day, 100 mg. the second day, and 150 mg. daily thereafter for fifteen to twenty doses. This is usually sufficient to alleviate pain. Daily treatments can be given only when the patient has normal renal function and no Bence-Jones proteinuria. In the presence of signs of renal damage the dose is given every other day. Weekly determination of blood urea nitrogen is required. The diet should be poor in animal protein.

Roentgen therapy may be helpful after the termination of the stilbamidine injections. Eighty per cent of the cases were relieved temporarily of pain. The disease is at best halted temporarily and relapses are frequent. Bence-Jones proteinuria and increase of globulin in the serum are not influenced. Stilbamidine appears to have a specific affinity for the abnormal nucleoproteins of the myeloma cells.—*Robert K. Arbuckle, M.D.*

Kelly, James F., Dowell, D. Arnold, and Downing, John E. Roentgen rays in prevention and treatment of infections. *Radiology*, Sept., 1948, *51*, 341–349.

The authors review the theory and clinical effects of roentgen treatment of infections and report 2 cases.

A seven year old girl developed tetanus five weeks after a foot injury. Roentgen therapy was started on the third day as there had been no response to antitetanus serum, penicillin, and streptomycin. The patient was free of evidence of toxemia four days after irradiation was started. Details of treatment are not given. The authors recommend roentgen therapy for tetanus in conjunction with other measures.

A case of gas gangrene involving the arm was given roentgen therapy starting the fourth day. Continued use of penicillin and anti-gas gangrene serum and repeated surgical removal of damaged tissue failed to prevent or control the gas infection. Not until roentgen therapy had time to take effect was improvement noted. Much infected tissue was never removed, but fully recovered under roentgen therapy.

The authors insist that tissues which may seem hopelessly diseased during the acute toxic phase often recover. They are not convinced that penicillin is of any value and consider serum unnecessary if roentgen radiation is used.—*Arthur A. Brewer, M.D.*

Riebeling, Manuel. Roentgen treatment of external infections due to bacillus anthracis. *Radiology*, Sept., 1948, *51*, 333–340.

Thirty-six patients with infections produced by *Bacillus anthracis* (demonstrated in smears in all cases) were treated with fractionated small doses of radiation of medium penetration. The factors as reported were: 140 kv., with filtration of "2.0 mm. aluminum to 0.25 mm. copper and aluminum," target-skin distance, 30 cm. Most cases were treated every 24 to 48 hours, the dose per field varying from 18 to 85 r. Treatment was "highly individualized."

Detailed case histories of 15 cases are reported. In all cases there was a prompt response to irradiation. *B. anthracis* disappeared from the exudate from the primary lesion and general and local manifestations subsided. No complications incident to treatment were observed.

Roentgen therapy appears to be of little significance for the mild localized infections, in many of which spontaneous recovery takes place. In cases with severe local or regional manifestations, such as those reported, the author believes the situation is quite different, and reports rapid and sometimes dramatic improvement in both the local and general condition.—*Chauncey N. Borman, M.D.*

Boden, Geoffrey. Radiation myelitis of the cervical spinal cord. *Brit. J. Radiol.*, Sept., 1948, *21*, 464–469.

The roentgen treatment of 161 patients with various lesions in the neck necessitated inclusion of the cervical portion of the spinal cord in the field, and of these patients 10 developed transient or progressive cervical myelitis. The patients fell into two groups: 145 were treated with large fields, resulting in homogeneous dosage to the whole cervical area; the other group of 16 were treated by small field, beam-directed therapy. The first group were lymphomas, aplastic carcinomas of the nose, mouth, throat or cervical lymph nodes, or inoperable thyroid carcinomas. Small fields were used for carcinoma of the posterior pharynx in the region of the arytenoids, or in the posterior pharyngeal wall.

Four patients who developed transient myelitis showed subjective evidences only. When

physical signs developed, the lesion was progressive and the signs were those of partial transection of the cord. In these cases, disease of the cervical vertebrae, intra- and extramedullary cord lesions, and cord lesions were all carefully excluded but autopsy control was obtained in only two. Of the patients treated with large fields, 6 developed myelitis, 3 with permanent changes; in the other group of 16 treated with directed beams there were 4, three of which had permanent cord involvement. In each case where progressive lesions developed, it was found that a larger dose than intended had been given. One case had had a previous unknown amount of irradiation; in a second, the side of an equilateral triangle had been taken as the height; in another, there was spread of the beams at the margins of adjacent fields; and, in another, the treatment had been administered at a greater focus-skin distance than had been calculated.

Calculation of the dose to the center of the cord was carried out subsequently and the estimated upper limit of safe treatment to the cord is somewhat below 3,500 roentgens in seventeen days when large fields are being used. With small fields, the small number of patients prevents firm conclusions, but 5,000 to 5,500 roentgens in seventeen days, or 2,000 roentgens in one day will cause damage in a proportion of cases.—*E. F. Lang, M.D.*

SCHENCK, S. G. Radiation therapy for Wilms' tumor of the kidney. *J. Pediat.*, Dec., 1948, *33*, 734–738.

The author outlines the accepted method of treatment of Wilms' tumors as follows:

1. Operation alone. This method is worthless in the presence of metastases or when the tumor is too large.

2. Operation and postoperative irradiation. This method has been the treatment of choice.

3. Preoperative roentgen irradiation. This method may make an inoperable tumor operable and minimize dissemination.

4. Preoperative and postoperative irradiation. This is the present accepted method by a majority of workers in this field. This method is at least the method of choice in the presence of metastases where it is used for palliation alone.

The usual roentgen technique, where a cure is possible, employs three portal, anterior, posterior, and lateral. The total dosage should be from 3,600 to 6,000 roentgens, treating one portal daily. The total daily dose should not exceed 200 r. In cases of massive tumor six smaller portals may be used. Operation should be delayed for four to six weeks after completion of roentgen therapy. In the presence of metastases 800–1,200 r is given through three portals.

The author treated 17 patients from 1930–1947 with Wilms' tumor. All the cases died. Average survival rate was 6½ months. The statistics referable to the various forms of therapy were not conclusive as to the treatment of choice to be employed.—*Rolfe M. Harvey. M.D.*

ARNESON, A. N., STANBRO, WILLIAM W., and NOLAN, JAMES F. The use of multiple sources of radium within the uterus in the treatment of endometrial cancer. *Am. J. Obst. & Gynec.*, Jan., 1948, *55*, 64–78.

This report is a study of a group of patients with carcinoma of the fundus of the uterus. The authors compare the results of treatment with gamma irradiation, using multiple weak radium capsules packed individually into the uterine cavity with the results obtained by the use of strong capsules placed in tandem in the uterine fundus.

In their group of 93 patients 45 were considered inoperable due to advanced cancer or some other lesion. Those patients who were treated by irradiation alone show a five year survival rate of 27 per cent, while those given preoperative roentgen therapy and intrauterine radium followed by hysterectomy show a 68 per cent survival for five years. Surgery alone was used in 18 patients of whom 84 per cent are alive and well.

The technique of multiple weak capsules of radium, each capsule containing approximately 6 mg. per running centimeter, is described by the author. While their statistics are non-convincing due to the limited number of patients treated, theoretically this method should give a better distribution of irradiation and, as indicated in the authors' postoperative specimens, there is less local necrosis with better distribution of the carcinocidal gamma rays. The authors find an improvement in their clinical results when using the multiple capsules as compared to the tandem position for the intrauterine radium. However, other factors also influence their clinical results. These are the histopathological type of the tumor, the biological properties of the tumor, and the size of the uterus.—*George W. Chamberlin, M.D.*

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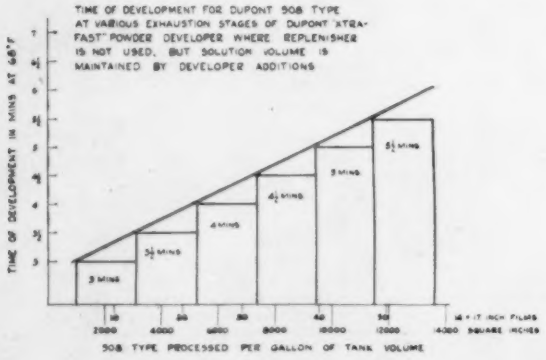
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TIME OF DEVELOPMENT IN MINS AT 68°F
3 MINS
3½ MINS
4 MINS
4½ MINS
5 MINS
5½ MINS
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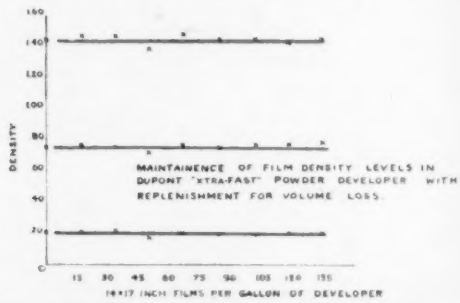
DENSITY
MAINTAINENCE OF FILM DENSITY LEVELS IN DUPONT "XTRA-FAST" POWDER DEVELOPER WITH REPLENISHMENT FOR VOLUME LOSS.
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plenisher. Chart II above shows how Replenishe maintains film density at a constant level.

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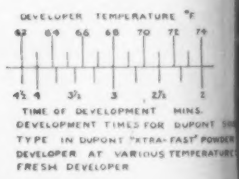

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